ENT Scholar

Volume 1 Issue 2 2012

Editor: Dr U Venkatesan
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Editor:

Balasubramanian Thiagarajan

ENT SCHOLAR

Volume 1 Issue 2 2012

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Tips and Tricks

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Balasubramanian Thiagarajan

Preventing nerve damage during thyroid surgeries

Introduction:

Thyroid gland is intimately related to two important nerves that control voice; i.e. Superior laryngeal and recurrent laryngeal nerves. Surgeon who operates on thryoid should always keep in mind the unpleasant morbidity caused by inadvertant injury to these nerves during surgery. Thyroid surgery is one of the commonly performed surgical procedures these days [1].

"*Can the thyroid*

gland when in the state of enlargement be removed…? If a surgeon should be so foolhardy

as to undertake it. .every step he takes will be environed with difficulty, every stroke of his

knife will be followed by a torrent of blood and lucky it would be for him if his victim lives

long enough to enable him to finish his horrid butchery. No honest and sensible surgeon

would ever engage in it" Samuel Gross 1848. Things have moved a long way since the famous quote of Gross.

With the common availability of state of the art haemostats like bipolar cautery, Radiofrequency cautery and harmonic scalpels more and more surgeons are emboldened to venture into this field. It is slowly becoming a border zone where surgeons of various specialities attempt to transgress (General surgeons, otolaryngologists and surgical endocrinologists). Currently available state of the art cautery devices like LigaSure[3]/ Harmonic scalpel [2]have really made surgeons job in securing haemostasis during this surgery real easy. Lot of credit should go to Theodar Kocher who showed that morbidity / mortality can be significantly reduced if meticulous dissection and precise ligation of blood vessels is carried out. Under his hands the mortality rate came down to less than 1%. He was awarded the Nobel Prize in recognition to his contribution to the knowledge of thyroid gland. After him it was left to Billroth to carry the torch of knowledge further.

Causes of Increased morbidity and mortality during thyroid surgeries:

Haemorrhage

Asphyxia

Air embolism

Infections

Almost all of these have been conquered by innovations in the field of anaesthesiology and surgical instrumentation. Excellent exposure and meticulous haemostasis will go a long way in helping to reduce the complications of thryoid surgery. It is imperative on the part of the surgeon to recognize the potential complications of this surgical procedure and take adequate steps to reduce their incidence in the interest of the patient. Blood less field will help the surgeon to identify vital structures like recurrent laryngeal nerves, parathyroid, and superior laryngeal nerves.

Commonly involved nerves during thyroid surgeries include:

Recurrent laryngeal nerves

Superior laryngeal nerves

Recurrent laryngeal nerves are closely related to the inferior vascular pedicle of thyroid gland (inferior thyroid artery) and superior laryngeal nerves are related to the superior vascular pedicle i.e. superior thyroid vessels. Injuries involving recurrent laryngeal nerves are more sinister in nature and can cause morbidities ranging from aspiration to stridor. Best way to avoid injuries to recurrent laryngeal nerve (more important) of the two is to identify the nerve in all cases[4,5].

Recurrent laryngeal nerve injury:

Recurrent laryngeal nerve injuries are more common in thyroid surgeries performed for:

Thyroid carcinoma

Toxic goitre – Due to increased vascularity which obscures the nerve due to excessive bleeding

Recurrent goitre – Due to adhesions and anatomical displacements

Clinical features of recurrent laryngeal nerve injuries [6]:

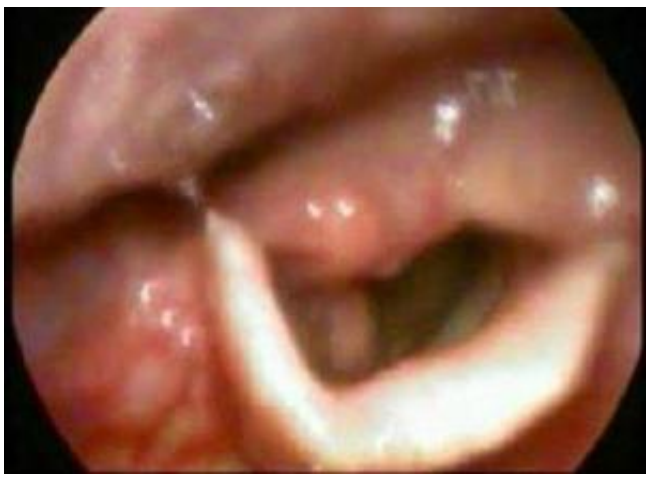


Image showing left vocal cord paralysis following injury to left recurrent laryngeal nerve

Unilateral recurrent laryngeal nerve injury:

Is the most common situation encountered. Left cord is affected commonly than the right as the left vagus nerve takes a more tortuous course. To start with the voice is breathy, but the normal vocal cord starts to compensate soon. The air way is adequate and there is no stridor in these patients. On indirect laryngoscopic examination the affected cord could assume any of the 6 positions described above. The cord may appear not to move, while the opposite cord will compensate for the lack of mobility.

These patients have a breathy voice. The breathiness of voice is caused by glottic chink which allows air to escape when the patient attempts to speak. Normal voice production is dependent on proper glottal closure resulting from bilateral adduction of the vocal cords. This adduction of vocal folds combined with subglottic air pressure causes the vocal folds to vibrate causing phonation.

Bilateral recurrent laryngeal nerve injury:

This is the most dreaded complication of thyroid surgeries. These patients manifest with stridor and tracheostomy need to be performed in order to secure the airway. This is commonly seen as a sequele to total thyroidectomy. Voice is normal in these patients.

Tips for avoiding injury to recurrent laryngeal nerve during thyroid surgery:

1. Detailed anatomical knowledge of recurrent laryngeal nerve and its varying relationships with that of inferior thyroid artery

2. Temptation to rmass ligate the pedicles (inferior) especially should be resisted

3. Ligatures should stay as close to the thyroid gland as possible always

4. Recurrent laryngeal nerve should be identified before securing inferior thryoid vessels

5. Haemostasis should be meticulously maintained at all times in order to provide good surgical field for identifying the nerve

6. Indirect laryngoscopy should always be performed before surgery in these patients to know the preoperative vocal cord status

7. First time is the best time for surgery. Always complete / ensure complete removal of the disease the very first time. Attempts at revision surgery is always fraught with dangers to the recurrent laryngeal nerve.

8. Common site of injury to recurrent laryngeal nerve is close to the Berry's ligament. This can be due to excessive traction, nerve getting caught within ligatures, nerve being injured due to electocoagulation.

9. A branched recurrent laryngeal nerve is more prone for injury during surgery

Role of inferior thryoid artery in identifying recurrent laryngeal nerve:

The inferior thyroid artery and its branches are intimately associated with the recurrent laryngeal nerve. This relationship is at the junction of middle and lower third of thyroid gland. The left recurrent laryngeal nerve ascends at a depth of tracheo oesophageal groove or slightly lateral to it at the lower pole of thyroid gland. The nerve on the left side crosses deep to the inferior thyroid artery/inbetween its terminal branches. It is rarely seen in a plane superficial to the artery.

The right recurrent laryngeal nerve is somewhat lateral in position at the lower pole of the thyroid gland. It courses more obliquely. This is a very common area of injury to right recurrent laryngeal nerve during thyroid surgery. Innumerable number of varying relationships with inferior thyroid artery has been described [7]. It is hence prudent to look for the nerve under the artery rather than supericial to it as this scenario is very rare.

Common relationship of recurrent laryngeal nerve to inferior thyroid artery [8]:

The recurrent laryngeal nerve has significant but varying relationship with the inferior thryoid artery.

On the left side, the recurrent laryngeal nerve passes behind the inferior thyroid artery in 50% of the cases and anterior to the artery in 20% of cases and may lie in between the branches of the inferior thyroid artery in 30% of cases. On the right side since the recurrent laryngeal nerve approaches the traceoesophageal groove more laterally, these relations are different on the right side. In half of the cases the recurrent laryngeal nerve passes between the distal branches of the inferior thyroid artery, in 30% of patients it may lie anterior to the artery, and in 20% of cases it may lie deep to the inferior thyroid artery.

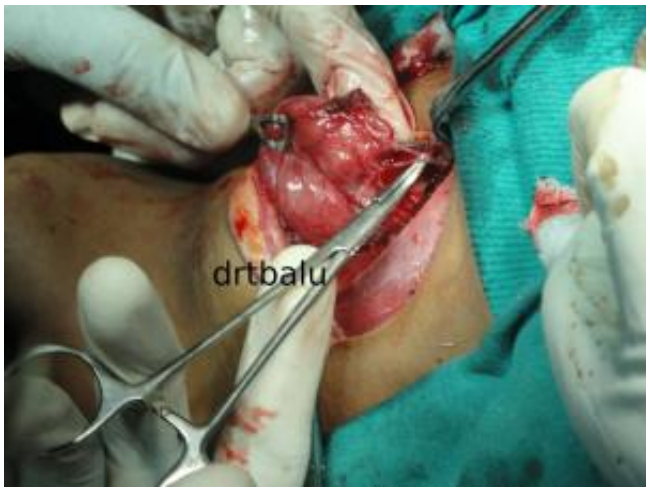


Figure showing Inferior thyroid vessels

Identification of inferior thyroid artery, careful ligation of all its branches close to the gland rather than away from it is very helpful in preventing damage to recurrent laryngeal nerve and inferior parathyroid glands. In rare cases the nerve can branch below the inferior thyroid artery and in this scenario it is safe for the surgeon to assume all these branches to be motor branches to the larynx and take extra care to avoid damage to them. Inferior thyroid artery (a branch from the thyrocervical trunk) appears from beneath the carotid sheath only when the thyroid gland is retracted medially and the jugular vein retracted laterally. This maneuver puts strain in the artery and helps in better visualization. Before entering the thyroid gland it divides into three branches inferior, posterior and internal. This artery also supplies the inferior parathyroid gland.

Relationship of recurrent laryngeal nerve to Berry's ligament:

Also known as suspensory ligament of Berry. This ligament attaches the postero medial aspect of thyroid gland to the sides of cricoid cartilage and first two tracheal rings. It is this very attachment that is responsible for the up and down mobility of thyroid gland which occurs during swallowing.

This is a rather crucial area. The recurrent laryngeal nerve is embedded close to the posterior portion of Berry's ligament and is prone for injury when this ligament is sectioned in order to free the gland from its attachment. Inferior laryngeal artery lies posterior to recurrent laryngeal nerve in this area. Bleeders form Berry's ligament should not be clamped blindly before identifying recurrent laryngeal nerve.

At the level of middle third of thyroid gland the recurrent laryngeal nerve is situated close to the capsule of the gland. In cases with pathological enlargement of thyroid glands this nerve may be enclosed within the thyroid capsule itself before entering the larynx. It is more prone for injury in large swellings involving thyroid gland.

Medial retraction of thyroid lobe makes the nerve more vulnerable during thyroid surgeries. This maneuver stretches the inferior thyroid artery and its branches displacing the nerve anteriorly in the

tracheo oesophageal groove exposing it to danger.

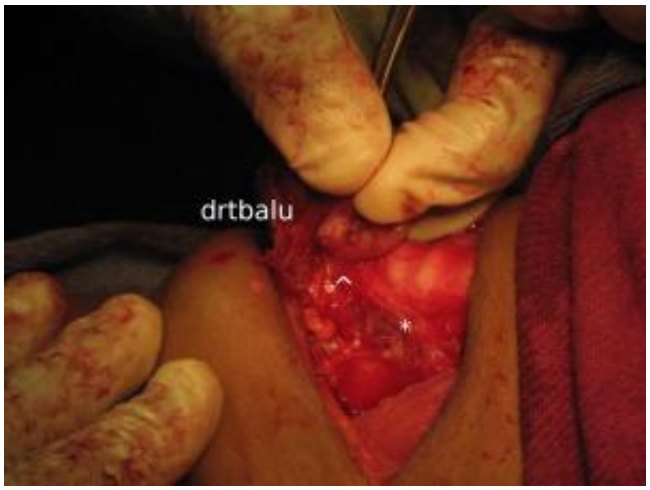


Figure showing Berry's ligament and recurrent laryngeal nerve: ^ – Berry's ligament * – Recurrent laryngeal nerve

Cricoarytenoid joint as a marker for the location of recurrent laryngeal nerve:

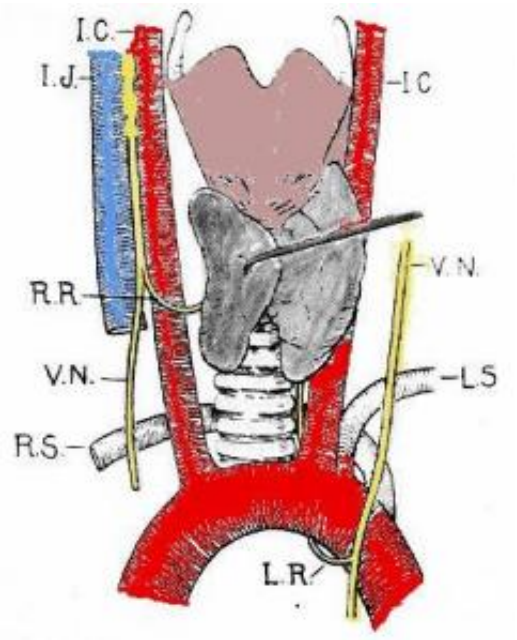
The recurrent laryngeal nerve enters the larynx deep to the inferior constrictor muscle and posterior to the cricoarytenoid joint. Inside the larynx it divides into a sensory and motor branches. The anteriorly directed motor branch is made up of 1000 axons. About 250 of the axons innervate the cricoarytenoid muscle, since it is the sole abductor of the vocal fold. The trachea, oesophagus and pyriform sinuses receive their sensory fibers from the posterior division of the recurrent laryngeal nerve before entering the larynx.

Recurrent laryngeal nerve can also be damaged if its blood supply is compromised during surgery. The blood supply to the recurrent laryngeal nerve comes from the inferior thyroid artery. The feeding branches are usually anterior to the nerve. Distally, the inferior laryngeal artery, a terminal branch of the inferior thyroid artery, supply the recurrent laryngeal nerve. It is always prudent to ligate the inferior thyroid artery closer to the gland after it has given off the branch to the recurrent laryngeal nerve.

Always consider non recurrent laryngeal nerve:

Non recurrent laryngeal nerve arises directly from vagus nerve in the neck. Hence it is not found in the usual position (i.e. Close to the inferior thyroid artery). Non recurrent laryngeal nerve is a very rare anamoly more common on the right side (0.5-0.6%) [9]. It is extremely rare on the left side (0.004%). At present there is no way of identifying this anamoly preoperatively with acceptable degree of accuracy. If CT scan neck shows retro oesophageal subclavian artery then this condition should be suspected [10].

Image showing Non recurrent laryngeal nerve



Use of operating loupe:

Operating loupes with atleast 4 times magnification with a good working distance is a real boon to the head and neck surgeon while performing thyroid surgeries. Routine use of operating loupes will minimize risk to the recurrent laryngeal nerve during thryoid surgeries.

Figure showing operating loupe

Superior laryngeal nerve [11]:

This nerve is also prone for injury during thyroid surgeries.

Anatomically superior laryngeal nerve is one of the branches of vagus nerve. Paralysis involving this nerve is frequently overlooked because of complex clinical picture. Functionally speaking the superior laryngeal nerve function can be divided into sensory and motor components. The sensori function provides a variety of afferent signals from supraglottic larynx. Motor function involves motor supply to ipsilateral cricothyroid muscle.

Role of cricothyroid muscle on phonation:

Contraction of cricothyroid muscle tilts the cricoid lamina backward at the cricothyorid joint causing lengthening, tensing and adduction of vocal folds causing an increase in the pitch of the voice generated.

Diagnosis of superior laryngeal nerve paralysis is based largely on symptomatology and clinical suspicion.

Symptoms:

1. Raspy voice

2. Voice fatigue

3. Volume deficit

4. Loss of singing volume

Kierner classified the superior laryngeal nerve into 4 types depending on the relationship of its external branch to the superior pole of thyroid gland.

Type I nerve: In this type the external branch of superior laryngeal nerve crosses the superior thyroid artery about 1cm above the superior pole of thyroid gland.

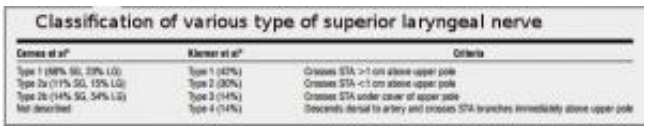
Type II nerve: In this type the external branch of superior laryngeal nerve crosses the superior thyroid artery within 1 cm of the superior pole of thyroid gland.

Type III nerve: In this type the external branch of superior laryngeal nerve crosses the superior thyroid artery under cover of the superior pole of thyroid gland.

Type IV nerve: In this type the external branch of superior laryngeal nerve descends dorsal to the superior thyroid artery and crosses its branches just superior to the upper pole of thyroid gland.

Awareness of these anatomical variations will help the surgeon in preserving this branch during head and neck surgeries.

Classification of various anatomical types of superior division of external laryngeal nerve:


Classification of various type of superior laryngeal nerve

Type 1 (68% SG, 39% LG)	Type 1 (42%)	Crosses STA >1 cm above upper pole
Type 2a (11% SG, 15% LG)	Type 2 (30%)	Crosses STA <1 cm above upper pole
Type 2b (14% SG, 54% LG)	Type 3 (14%)	Crosses STA under cover of upper pole
Not described	Type 4 (14%)	Descends dorsal to artery and crosses STA branches immediately above upper pole

Superior laryngeal nerve is highly vulnerable during ligation of superior pedicle of thyroid gland. Routine identification of this nerve is rather difficult without dissecting through pharyngeal constrictors. In nearly 20% of cases it is not located close to the ligation point of superior pole of thyroid at all. Hence routine identification of this nerve during thyroid surgery has not been advocated. It is safe to ligate superior thyroid artery as close to the superior pole of thyroid gland as possible. It is infact safer to identify the brances of superior thyroid artery and avoid ligating the main trunk as in majority of cases superior laryngeal nerve lies rather close to the main trunk.

Use of nerve stimualtors:

Eventhough nerve monitors and stimulators have been advocated their usefulness still remains highly questionable. One study reports that they were able use it only to identify superior laryngeal nerve. It did not actually aid in the anatomical dissection of recurrent laryngeal nerve [12].

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Septal Hematoma?

Nasal deformities are just around the corner

March 16, 2012 · Rhinology

Authors

Balasubramanian Thiagarajan

Abstract

Septal hematoma is accumulation of blood in the subperichondrial compartment of nasal septum. It is commonly caused by injuries involving the nose and face. Unilateral septal hematoma commonly heals causing fibrosis to occur between the septal cartilage and the perichondrium. Bilateral septal hematoma compromises blood supply to nasal septum causing liquefaction necrosis of septal cartilage. This ultimately leads to depressed dorsum of nose.

Septal Hematoma

Introduction:

Septal hematoma is a collection of blood under the nasal septal cartilage [1]. Since nose is the most prominent portion of face it is prone for injuries leading on to septal hematoma formation. Even though incidence of septal hematoma is rather rare early diagnosis and management of this condition will go a long way in preventing attendant complications like septal abscess, septal perforation and saddle nose deformity [2]. Injuries involving the anterior portion of nasal septum commonly causes septal hematoma [3].

Pathophysiology:

When the nasal septum is subjected to sharp buckling stress, the submucosal blood vessels are frequently damaged, and if the mucosa remain intact, will result in the formation of hematoma. If the trauma is severe enough to fracture the septal cartilage, the blood will seep to the opposite side causing bilateral septal hematoma. The nasal septum is usually 2-4 mm thick, hence it is common to see fractures involving nasal septum. This bilateral septal hematoma is dangerous because it compromises the nutrition of the septal cartilage the most and cause dissolution of the whole cartilagenous septum itself. Since the nutrition of the cartilage is dependent on the intact perichondrium, elevation of the perichondrium away from the cartilage causes necrosis of the cartilage. Avascular cartilage can remain viable only for 3 days after compromise of the perichondrium. Cartilage absorption can occur with alarming rapidity.

If the hematoma is small and unilateral it may not cause necrosis of the cartilage, but may be absorbed causing permanent thickening of the nasal septum and gross fibrosis. In adults septal hematoma occurs following facial trauma / nasal bone fracture. In children even trivial fall / injury can cause septal hematoma [5].

Signs & Symptoms: usually occur within the first 24-72 hours [4].

1. Nasal obstruction: This is the dominant symptom. In unilateral septal hematoma nasal obstruction is unilateral, and in bilateral septal hematoma nasal obstruction is bilateral.

2. Pain

3. Rhinorrhoea

4. Fever

On examination:

Presence of smooth swelling in the nasal septal area. Either on one side or on both sides. In unilateral septal hematoma, there is asymmetry of nasal septum. Direct palpation of nasal septum also will help in confirming the diagnosis of septal hematoma.

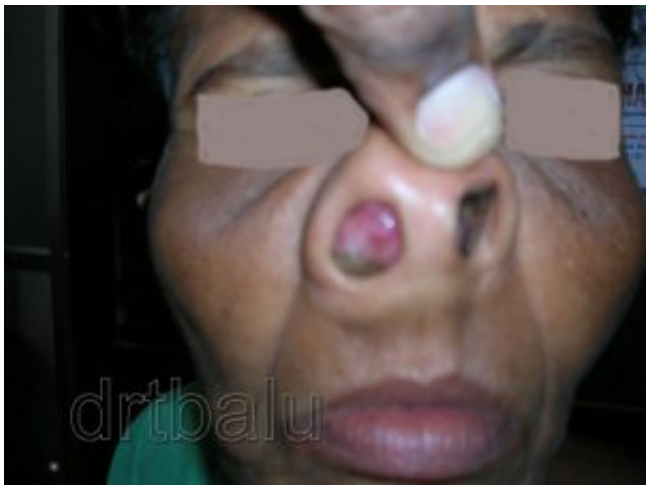

Fig. 1: Figure showing unilateral septal hematoma

Treatment:

Emergency drainage of septal hematoma is a must in these patients [6]. It has been shown that early surgical drainage of the hematoma reduces the risk of cartilage necrosis, and hence is always indicated. A hemitransfixation incision (incision made at the lower border of the nasal septal cartilage) is used, since the perichodrium is already lifted off the cartilage the acumulated blood and infected material is aspirated. The state of the cartilage is assesed and if there is any defect it is advisible to support the defect with homograft cartilage. These cartilage grafts can be used even if abscess formation has occured thus effectively preventing saddle nose deformities. The homograft cartilage can be harvested from patients who have under gone submucosal resection of the nasal septum. These harvested cartilages can be stored in 0.1% sodium mecurothiosalicylate.

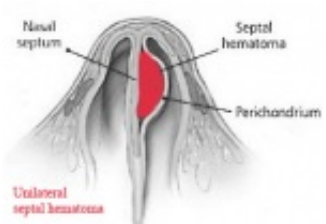

Fig. 2: Figure showing formation of septal hematoma

Incision site

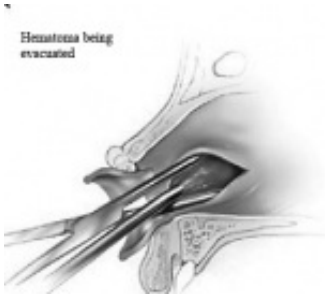

Figure showing septal hematoma being evacuated

Complications of septal hematoma:

External deformity of the nose: The cartilagenous dorsum of the nose is supported by the septal cartilage and if this is lost then dorsal saddling can occur causing pig snout deformity (Pig nose like). If this injury occur during childhood, it may also affect the development of the whole of the middle third of the face causing resultant maxillary hypoplasia.

Septal abscess: Hematoma is a good culture medium and hence may become infected causing abscess formation. This complication is always associated with severe pain, together with manifestations of toxemia, such as increased pulse rate.

Septal deviation:

Minor hematomas especially the unialteral ones may get absorbed and appear as thickened areas in the nasal septum with extensive fibrosis leading on to deviation of nasal septum to that side due to contracture caused by fibrosis.

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Huge rhinolith nasal cavity an interesting case report and a review of literature

May 5, 2012 · Rhinology

Authors

Balasubramanian Thiagarajan

Abstract

Rhinoliths are calcareous deposits (stone like) inside the nasal cavity. These stone like structures are highly friable and may crumble when crushed. This interesting case report discusses a patient with a huge rhinolith inside the nasal cavity. Rhinoliths since they crumble easily can be removed after crushing with a luc's forceps via the nasal cavity. Since the rhinolith in this patient was very large extending up to the choana it was removed via lateral rhinotomy approach in order to avoid excessive injury to nasal mucosa during the process of removal.

Huge rhinolith nasal cavity an interesting case report and a review of literature

Introduction:

Rhinoliths are also known as nasal calculi are calcareous deposits present inside the nasal cavity [1]. Rhinoliths are of two types: Exogenous and Endogenous.

Exogenous rhinolith: If concretions occur around a impacted foreign body then it is considered to be exogenous in nature. These calcareous deposits around intranasal foreign bodies is the most common variety of rhinolith [2].

Endogenous rhinolith: If concretions occur around blood clot / inspissated foreign body then it is considered to be endogenous in nature.

This condition is commonly diagnosed by history and anterior rhinoscopy [3]. This condition is common in adults and elderly individuals. Unilateral foul smelling blood tinged nasal discharge in an adult should always raise suspicion of rhinolith. Since rhinoliths are commonly seen in the anterior nasal cavity, anterior rhinoscopic examination of nose clinches the diagnosis [4].

Patients with rhinolith usually present with:

1. Unilateral nasal obstruction

2. Unilateral foul smelling blood tinged nasal discharge

3. Hard mass inside the nasal cavity

Case Report:

60 years old male came with complaints of

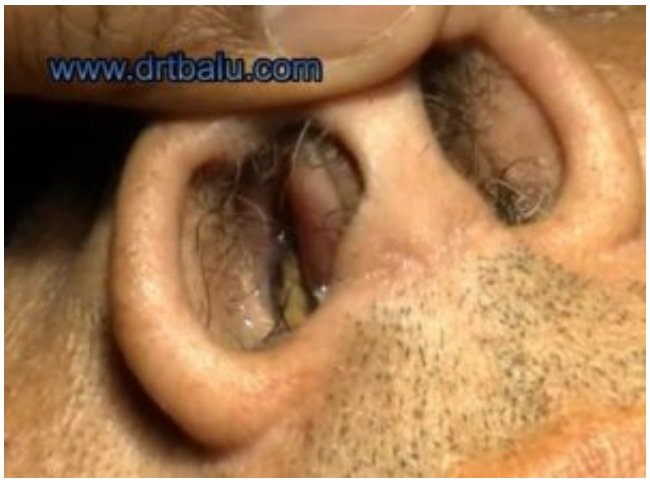
1. Right sided nasal block – 3 years

2. Foul smelling blood tinged discharge right nose – 3 years

3. Right sided head ache on and off – 4 years

Anterior rhinoscopy:

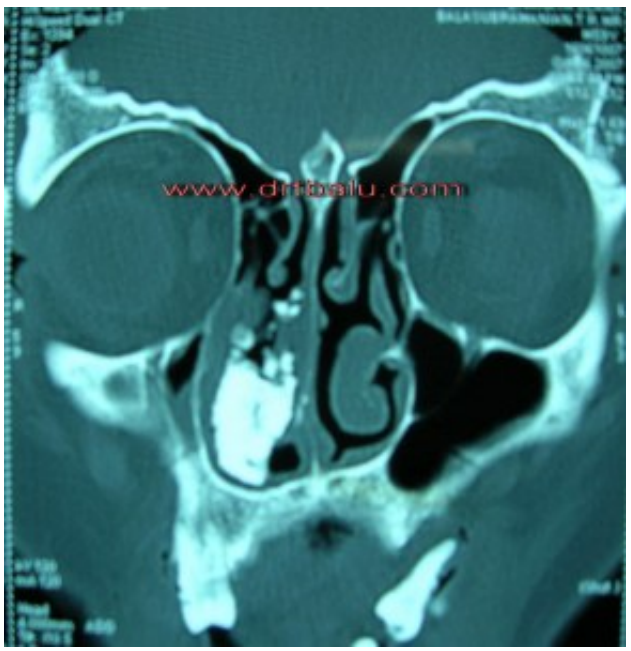
Dirty white irregular hard mass could be seen occupying the entire right nasal cavity. The same mass was found pushing the nasal septum to the left side. The mass was found to be gritty on probing. The probe could be passed all around the mass.



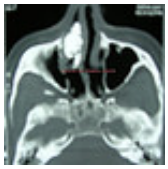
Anterior rhinoscopy showing rhinolith

CT scan:

Axial and coronal CT scan showed radio opaque irregular mass occupying the entire right nasal cavity.



Coronal CT scan of nose and sinuses showing rhinolith



Axial CT of nose and sinuses showing rhinolith

Management:

Since the mass was quite large and was extending up to the posterior end of middle turbinate it was decided to remove it using lateral rhinotomy approach in order to prevent damage to nasal mucosa. A pervia naturalis approach was not considered because the mass was considerably hard (not friable) and was large.

Under general anesthesia, using Moore's lateral rhinotomy approach the nasal cavity was entered. The mass was removed completely and the wound was closed in layers.

Discussion:

The term rhinolith is derived from Greek (rhino – nose lithos – stone). It is considered to be a rather rare condition i.e. About 1 in 10,000 otolaryngology patients [5]. It was Bertholin who first gave the accurate description of this condition in 1654 [2]. Rhinoliths are usually irregular brownish / grey colored masses present in the anterior portion of the nasal cavity.

For some unknown reason males seem to be commonly affected than females [3]. The exact pathogenesis involved in the development of rhinolith is still not known. It has been suggested that impacted foreign body / mucous plugs / blood clot may incite inflammatory reaction and stimulate deposition of minerals and salts. The salts which gets deposited around the nidus is derived from nasal secretions, tear and inflammatory exudate [1]. The nidus of rhinolith is usually a foreign body [5]. Even gauze swabs inadvertently left inside the nasal cavity following surgery has been known to cause rhinolith. Radiology is usually diagnostic. Typical radiological picture is radio opacity with sometimes central opacity. The central radiolucency could be due to the presence of organic material which could have formed the nidus for rhinolith. This description was first given by Mac Intyre[6]in 1900. CT scan usually cannot differentiate rhinolith from other calcified masses.

Differential diagnosis of rhinolith include:

Hemangioma

Osteoma

Calcified polyp

Chondroma

Osteosarcoma

Conclusion:

This case is being presented because of its large size, lack of friability and the surgical approach which was resorted to inorder to remove it. Lateral rhinotomy approach was resorted to inorder to prevent mucosal damage which could occur if removal is attempted pervia naturalis.

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Nasal polyposis in children

A review article

January 17, 2012 · Rhinology

Authors

Balasubramanian Thiagarajan

Abstract

This article discusses various causative factors of nasal polyposis in children. It is a review of literature on this subject, supplemented by the author's personal experience. Even though nasal polyposis is rather uncommon in children, when present they should be throughly investigated to rule out other sinister lesions. Imaging has a vital role to play in diagnosis of these patients. Antrochonal polyp is currently the commonest nasal polyp seen in children.

Funding Statement

This article was not funded by any agency

Nasal polyposis in children

Introduction:

Studies reveal that eventhough nasal obstruction and discharge are common in children, nasal polyposis [1] is rather uncommon. Majority of nasal polypi in children are caused by infection and inflammation of nasal / sinus mucosal lining. Among the types of nasal polyposis seen in children about 1/3 of these patients have antrochoanal polyp [2].

Causes of nasal polyposis in children:

Antrochoanal polyp

Inflammatory polyp

Polyp due to cystic fibrosis

Antrochoanal polyp:

Synonyms: Antrochoanal polyp, Killian's polyp [3], Nasal polyp.

Palfyn [4] described the first case of antrochoanal polyp 1n 1753. Since he found the polyp filling the nasopharynx and extending below uvula he thought that it could have arisen from the choana. Killian in 1906 demonstrated that this polyp arose from maxillary sinus antrum. According to Stammberger 70% of antrochoanal polyp exited out of the maxillary sinus antrum via the accessory ostium [5].

Definition [6]:

Antrochoanal polyp is a benign solitary polypoidal lesion arising from the maxillary sinus antrum causing opacification and enlargement of antrum radiologically without any evidence of bone destruction. It eixts the antrum through the accessory ostium reaches the nasal cavity, expands

posteriorly to exit through the choana into the post nasal space.

Incidence:

It commonly affects young children and adolescents.

Etiopathogenesis:

Antrochoanal polyp is said to originate in the maxillary antrum due to inflammation [7]. This condition has been commonly documented only in non atopic persons [6]. Its etiology is still unknown.

Various theories have been proposed to account for the pathogenesis of this disorder:

Proetz theory[4]:

Proetz suggested that this disease could be due to faulty development of the maxillary sinus ostium, since it was always been found to be large in these patients. Hypertrophic mucosa of maxillary antrum sprouts out through this enlarged maxillary sinus ostium to get into the nasal cavity. The growth of the polyp is due to impediment to the venous return from the polyp. This impediment occur at the level of the maxillary sinus ostium. This venous stasis increases the oedema of the

polypoid mucosa thereby increasing its size.

Bernoulli's phenomenon: Pressure drop next to a constriction causes a suction effect pulling the sinus mucosa into the nose. According to this theory there is a pressure drop at the level of infundibular area causing a relative negative pressure. This negative pressure is sufficient to cause prolapse of maxillary antral mucosa into the nasal cavity. This prolapsed mucosal lining begins to enlarge in size due to oedematous reaction causing formation of polypoidal tissue.

Mucopolysaccharide changes: Jakson postulated that changes in mucopolysaccharides of the ground substance could cause nasal polyp. These changes lead to water retention within the submucosal compartment could lead to polyp formation.

Mill's theory:

Mills postulated that antrochonal polyp could be maxillary mucoceles which could be caused due to obstruction of mucinous glands.

Ewing's theory: Ewings suggested that an anomaly which could occur during maxillary sinus

development could leave a mucosal fold close to the ostium. This fold could later be aspirated into the sinus cavity due to the effects of inspired air causing the development of antrochonal polyp.

Vasomotor imbalance: This theory attributes polyp formation due to autonomic imbalance.

Infections: Recurrent nasal infections have also been postulated as the cause for nasal polyp. This theory suggests that acinous mucous glands within the maxillary sinus cavity gets blocked due to infection / inflammtion involving the mucous lining of the sinus cavity. This leads to the formation of a cystic lesion within the maxillary sinus cavity. This cyst gradually enlarges to occupy the whole of the maxillary sinus cavity. It exits the sinus cavity by enlarging the accessory ostium and enters the nasal cavity. Usually these cysts arise from the antero inferior / medial wall of maxillary

antrum. Macroscopically the portion of A/C polyp within the maxillary antrum is cystic in nature, while the component that has prolapsed via the accessory ostium is solid in nature.

Possible reasons for posterior migration of antrochoanal polyp:

Classically antrochonal polyp presents posteriorly. The polyp could be clearly seen occluding the post nasal space. Possible reasons for this posterior presentation include [6]:

1. The accessory ostium through which the polyp gets out of the maxillary antrum is

present posteriorly.

2. The inspiratory air current is more powerful than the expiratory air current thereby

pushes the polyp posteriorly.

3. The natural slope of the nasal cavity is directed posteriorly, hence the polyp

always slips posteriorly.

4. The cilia of the ciliated columnar epithelial cells lining the nasal cavity always

beats anteroposteriorly pushing the polyp behind.

Histology:

Shows respiratory epithelium over normal basement membrane. The interstitial layer is grossly oedematous, with no eosinophils. The interstial layer contains other inflammatory cells.

Clinical features:

This disorder is commonly unilateral. Bilateral antrochoanal polyp is very rare condition. Only a few handful of such cases have been reported in literature so far.

1. Unilateral nasal obstruction

2. Unilateral nasal discharge

3. Headache (mostly unilateral)

4. Epistaxis

5. Sleep apnoea

6. Rhinolalia clausa due to presence of polyp in the post nasal space

7. Difficulty in swallowing if the polyp extends into the oropharynx

Fig. 1: Antrochoanal polyp

This is an endoscopic image of antrochoanal polyp Anterior rhinoscopy may show the polyp as glistening polypoidal structures. They will be insensitive to touch. this feature helps to differentiate it from a hypertrophied nasal turbinate. Postnasal examination will show the polyp if extending posteriorly at the level of choana. If it fills up the nasopharynx it will be visible there. Xray paranasal sinuses will show a hazy mazillary antrum. CT scan of paranasal sinuses is diagnostic. It will show the polyp filling the maxillary antrum and exiting out through the accessory ostium into the nasal cavity. Coronal CT scan showing antrochoanal polyp Coronal CT Plain The antrochoanal polyp is dumb bell shaped with three components i.e. antral, nasaland nasopharyngeal.
Treatment: This is a surgical problem. Formerly it was treated by avulsion of the polyp transnasally. This method led to recurrences. A caldwel luc approach was preferred in patients with recurrences. In caldwel luc procedure

in addition to the polypectomy, the maxillary antrum is entered via the canine fossa and the antral component is completely excised. Endoscopic approach: With the advent of nasal endoscope this approach is the preferred one. Using an endoscope it is always easy to completely remove the polypoid tissue. The uncinate process must also be completely excised. Endoscopic approach has the advantage of a complete surgical excision with negligible recurrance rates. Antrochoanal polyp in the choana Endoscopic view of choanal portion of antrochoanal polyp Balasubramanian thiagarajan, drtbalu Creative commons Differences between antrochoanal polyp / Ethmoidal polyp

Antrochoanal polyp	Ethmoidal polyp
Solitary	Multiple
Arises from maxillary antrum	Arises from ethmoidal air cells
Has three components	Has only one component
Infection plays a role in its pathogenesis	Allergy is supposed to play a role
Common in adolescents	Common in adults / elderly

Coronal CT scan showing antrochoanal polyp arising from maxillary antrum exiting out of accessory ostium and entering the nasal cavity Balasubramanian thiagarajan, drtbalu Creative commons

Recent advances:

Current research involving Nitric oxide has thrown light into the possible etiopathogenic factors involved in the genesis of antrochonal polyp. Nitric oxide have been shown to play a major role in nonspecific immune reactions and inflammation in a variety of tissues. Endogenous nitric oxide is synthesized from Largenine by the effect of nitric oxide synthase. This all important nitric oxide

synthase exists in three forms:

1. Endothelial nitric oxide synthase

2. Neuronal nitric oxide synthase

3. Inducible nitric oxic synthase

Out of these three types the Inducible nitric oxide synthase has been detected not only in epithelium but also in macrophages, fibroblasts, neutrophils, endothelium and vascular smooth muscle.

Studies have revealed that antrochoanal polyp tissue contained more nitric oxide than normal tissues. Increased nitric oxide production could be from epithelial / inflammatory cells. Among inflammatory cells eosinophils play an important role in production of nitric oxide. Studies have also revealed that Inducible nitric oxide synthase play an important role in the pathogenesis of antrochonal polyp.

Sphenochoanal polyp is another rare unilateral nasal polyp that presents posteriorly occluding the choana. In fact this condition should be differentiated from antrochoanal polyp.

Cystic fibrosis:

This is another condition that can cause nasal polyposis in children. These children present with:

Pneumonia

Pancreatic insufficiency

Meconium ileus

Rectal prolapse

Biliary cirrhosis & portal hypertension

This genetic disorder is known to affect approximately 1 in 2500 live births. These patients have abnormal choloride transport, which is actually caused by defective chloride channel conductance which is actually regulated by cyclic AMP. This disorder is caused by mutation involving chromosome 7 which codes for chloride channel protein.

Attempts to seek evidence in adult patients with nasal polyposis for the presence of cystic fibrosis has not been fruitful [8].

The incidence of nasal polyposis in patients with cystic fibrosis ranges between 15 – 40%. This is a high variation considering the frequency of nasal polyposis in children [9]. Patients with cystic fibrosis invariably develop nasal polyposis after their 5th year or before they reach 20 years of age [10].

Studies performed by Toss et all have not demonstrated any morphological / histological differences between nasal polypoidal tissue between cystic fibrosis and non cystic fibrosis groups. This actually points towards the common underlying common pathogenesis.

Role of sweat test in the diagnosis of cystic fibrosis:

This is actually the gold standard test in the diagnosis of cystic fibrosis.

Sweat test is usually done in the forearm. It can also be done on the thighs.

Stimulation of sweat production:

This is actually the first step in sweat test. Electrodes containing pilocarpine is placed over the skin. Small current is passed through the electrodes so that pilocarpine will enter skin and stimuate secretion of sweat. This current is actually not painful but causes a tingling sensation. After about 10 minutes the electrodes are removed and a filter paper patch known as sweat patch is used to collect sweat. Chloride levels in sweat of patients with cystic fibrosis is supposed to be very high.

Seat chloride ranges:

Less than 30 = normal

30-59 = Borderline

60 and above is indicative of cystic fibrosis

Screening for the presence of AF508 gene could serve as a pointer for diagnosing cystic fibrosis.

Serum levels of immunoreactive trypsinogen has been found to be elevated in infants with cystic fibrosis.

Characteristic feature of sinusitis in these patients is the range of microbes that have been isolated by culturing the secretions. These organisms include: Psuedomonas aeruginosa, and staphylococcus

aureus.

Major nasal symptoms seen in these patients include:

Nasal block

Mucopurulent secretions

Head ache

Medical management has a very limited role to play in the managment of chronic sinusitis in patients with cystic fibrosis. Only role played by antibiotics in these children is to limit the damage due to repeated lower airway infections. Colonization of lower respiratory tract by pseudomonas is commonly seen in these patients. Nasal douching if preformed repeatedly will help in minimizing these colonies being formed in the lower airway.

Encephaloceles / Meningoceles:

These are congenital neural tube defects presenting as polypoidal masses inside the nasal cavity. It is imperative to differentiate these lesions from nasal polypi. These lesions can be identified by the presence of cough reflex. These masses change in size according to the phases of respiration.

High resolution CT scan images and MRI images helps in the diagnosis of this condition. After excision of these masses the defect in the skull base should be closed using a three layer graft. This will help in avoiding troublesome CSF leaks which are common in these patients following surgery.

Nasal polyposis associated with Primary ciliary dyskinesia:

This condition when associated with bronchiectasis and situs inversus totalis it is known as kartagener's syndrome. These patients have unrelenting nasal discharge without any symptom free interval. Since nasal mucosal ciliary beat is suboptimal in these patients saccharin clearance test will help in diagnosing this conditon.

Saccharin test:

This test is performed by placing a 1 mm diameter saccharine tablet just behind the anterior end of inferior tubinate / corresponding area of nasal septum. Patient is asked to sit quiety leaning forward. Patient is instructed not to sniff or attempt to clear the nose. The time taken for perception of saccharine taste after placement in the nasal cavity is recorded. Saccharine is dissolved in the mucous layer and is transported posteriorly to the nasopharynx by the nasal mucosal ciliary clearance mechanism. Average saccharine clearance time is 7 – 15 minutes. In patients with primary ciliary dyskinesia the clearance time could well be in excess of 1 hour.

FESS is useless in these patients, because ciliary mechanism is not going to become normal following surgery.

Allergic fungal sinusitis:

This is a non invasive disorder. Commonly caused by aspergillus infection. It is seen in immunocompetent individuals. These patients present with unilateral nasal polyposis with presence of greenish white crusts.

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Acknowledgements

Dr R Geetha for publishing this article

Malignant otitis externa a review of current literature

Difficult to diagnose and troublesome to treat

January 17, 2012 · Otology

Authors

Balasubramanian Thiagarajan

Abstract

Malignant otitis externa is a severe debilitating disorder that involves the external auditory canal. The term “Malignant Otitis Externa” is actually a misnomer. It has been coined to indicate the destructive capabilities of this disorder. This article discusses etiopathogenesis, diagnostic problems and various management modalities available to manage the same.

Funding Statement

This article was not funded by any agency.

Malignant otitis externa a review of current literature

[1]

Balasubramanian T

Introduction:

Malignant otitis externa is a inflammatory disorder involving the external auditory canal caused by pseudomonas organism. Majority of these patients are elderly diabetics. This condition is termed as malignant otitis externa because of its propensity to cause complications. Hence the term malignant must not be construed in a histological sense. This condition was first described by Meltzer and Kelemen in 1959[2]

It was Chandler in 1963 who coined the term “Malignant otitis externa” [3]. This condition commonly affects elderly diabetics who have decreased immunity. Studies reveal that it is more common among insulin dependent diabetics. Current literature also reports a few cases of Malignant otitis externa involving infants / young insulin dependent diabetics. The aim of otolaryngologist is to differentiate this condition from that of real malignancy i.e. Squamous cell carcinoma. Currently fluoroquinolones hold lots of promise in managing these patients.

History:

1838 – Toulmousch reported the first case of otitis externa

1959 – Meltzer reported a case of pseudomonas osteomyelitis of temporal bone

1968 – Chandler discussed the various clinical features and described it as a distinct clinical entity

Epidemiology:[4]

The typical patient with malignant otitis externa is an elderly diabetic, with males outnumbering females by twice the number. This could be due to the possibility of males being more prone to secrete wax which are more acidic in nature. Malignant otitis externa is very rare in children; if present it will be associated with malnutrition or HIV infection.

Pathophysiology:

Malignant otitis externa is known to affect the external auditory canal and temporal bone. The causative organism being pseudomonas aeruginosa. These patients are invariably elderly diabetics. This disorder usually begins as otitis externa and progresses to involve the temporal bone. Spread of this disease occurs through the fissures of Santorini and osteo cartilagenous junction. This disorder could be caused by a combination of poor immune response and peculiar characteristics of the offending microbe.

Immunity is reduced in patients with :

1. Diabetes mellitus

2. Blood cancer

3. HIV infections

4. Patients on anticancer drugs

Diabetic microangiopathy plays a vital role in the reduction of tissue perfusion causing opportunistic infections involving the area [5]. Rubin identified triggering factor for Malignant otitis externa in more than 60% of cases. He was able to elicit history of attempts at removing wax, use of ear buds etc [6].

It should also be remembered that diabetic patients have impaired phagocytosis, poor leukocytic response, and impaired intracellular digestion of bacteria. Diabetic patients secrete wax which has less lysozyme content than normal thereby reducing the effectiveness of wax as an antimicrobial agent.

Pseudomonas aeruginosa is a gram negative aerobe with polar flagella. It is found on the skin. It invariably behaves like an opportunistic pathogen. The pathogenicity of this organism is due to ability to secrete exotoxin and various enzymes like lecithinase, lipase, esterase, protease etc. Since this organism is clothed by a mucoid layer it is resistant to digestion by macrophages.

Clinical features :[1]

The patient gives history of trivial trauma to the ear often by ear buds, followed by pain and swelling involving the external auditory canal. Pain is often the common initial presentation. It is often severe, throbbing and worse during nights. It needs increasing doses of analgesics. On examination granulation tissue may be seen occupying the external canal. It often begins at the bony cartilaginous junction of the external canal. Discharge emanating from the external canal is scanty and foul smelling in nature. When the discharge is foul smelling it indicates the onset of osteomyelitis. Ironically the patient does not have fever or other constitutional symptoms.

Otoscopy: Reveals granulation tissue at the bony cartilaginous junction. The ear drum is usually normal. The external auditory canal skin is soggy and edematous.

Cranial nerve palsies are common when the disease affects the skull base. The facial nerve is the most common nerve affected. As the disease progresses the lower three cranial nerves are affected close to the jugular foramen.

Intracranial complications like meningitis and brain abscess are also known to occur.

Fig. 1: Facial palsy

Malignant otitis externa with lower motor neuron type facial palsy

Spread of infection:

Spread of infection:

1. Inferiorly through the stylomastoid foramen to involve the facial nerve.

2. Anteriorly to the parotid

3. Posteriorly to the mastoid and sigmoid sinus

4. Superiorly to the meninges and brain

5. Medially to the sphenoid

6. Spread through vascular channels are also common

Role of Imaging in diagnosis:

1. Conventional radiology is of no use in the diagnosis

2. CT scan [7]is useful in assessing bone involvement

3. MRI scan is useful in assessing soft tissue involvement

4. Radionucleotide scan using Technitium 99 helps in the diagnosis. This is really useful during the very early stages [4]of this disorder. Fixation of Technitium correlates with high degree of osteolytic activity which is commonly seen in these patients. This test is highly accurate 100% but its specificity is rather low [8]. Gallium-67 scintigraphy is very useful for prognostic evaluation because of its high specificity [9].

Levenson's criteria in diagnosing Malignant otitis externa :[1]

1. Refractory otitis externa

2. Severe nocturnal otalgia

3. Purulent otorrhoea

4. Granulation tissue in the external auditory canal

5. Growth of pseudomonas aeruginosa from external canal

6. Presence ofdiabetes/ Immunocompromised state

Radiological staging:

Grade	Diagnostic criteria
I	Disease limited to soft tissue not involving bone refractory to standard antibiotic therapy for more than 4 weeks
II	Earliest form of Malignant otitis externa with involvement of Mastoid bone
III	Malignant otitis externa extending medially to involve petrous portion of temporal bone
IV	MOE extending medially to involve the petrous apex or withcranial nerve involvement or pread anteriorly to involve thefacial bones, posteriorly to involve the occipital bone, orspread to the contralateral base of skull

Fig. 2: External canal granulation

External canal granulation in a patient with Malignant otitis externa **Staging and classification:**

Stage	Ga67	TC99	Extent of Disease
I	**+**	**-**	**Soft tissue(Necrotising Otitis)**
II	**+**	**+**	**Ear & Mastoid(Skull base osteomyelitis)**
III	**+**	**+**	**Extensive skull base osteomyelitis**

Management: Extensive surgical procedures have failed miserably to cure this condition. The role of surgery is confined to only exclusion of malignancy by biopsy. Wound debridement is a possibility in advanced cases. Medical management: Carbenicillin, Pipercillin, Ticarcillin can be used. Third and forth generation cephalosporins can be used. Ciprofloxacillin in doses of 1.5 g – 2.5 g /day in divided doses can be administered for a period of 2 weeks. Gentamycin can also be administered parenterally in doses of 80 mg iv two times a day in adults.

Acknowledgements

I acknowledge Dr R Geetha for publishing this article.

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Carhart's notch

Its implications

February 11, 2012 · *Otology*

Authors

Balasubramanian Thiagarajan

Abstract

Carhart's notch is classically seen as a dip centered around 2 kHz range of bone conduction curve audiometery. This feature is seen in patients with otosclerosis. This article attempts to discuss why this dip is caused in the bone conduction audiometry curve in these patients.

Carhart's notch

Introduction:

This is seen in bone conduction audiograms of patients with otosclerosis [1]. This is a dip at 2000 Hz in the bone conduction audiograms of these patients. Some authors consider this to be an artifact. After stapes surgery there is demonstrable over closure of air bone gap. There is also effective improvement in the patient's level of hearing at 2 KHz frequency levels.

Discussion:

Audiogram in airconduction shows a decrease in air conduction at all frequencies. Carharts notch [2]is actually a decrease in bone conduction of 10-15 dB seen around 2 kHz frequency. Bone conduction actually means sensorineural reserve. After successful stapes surgery the carharts notch disappears when the conductive hearing improves. This fact shows that carharts notch in no way represents sensorineural reserve of a patient. It is hence considered to be an arifact due to stapes fixation. This phenomenon was first described by Raymond Carhart in 1950. He attributed this phenomenon to stapes fixation. According to Tondroff carharts notch is not a true indication of cochlear reserve since it could be corrected by successful stapes surgery.

The frequency of resonance of middle ear has been identified as 800 – 1200 Hz [3]. Considering this to be a fact then one vital question about carhart's notch remain unanswered " Why is the dip seen at 2 kHz instead of 1200 Hz?" The answer to this question was provided by Zwislocki in 1957. He was able to demonstrate clearly that the primary resonance frequency for ossicular chain bone conduction falls between 1600 – 1700 Hz [4].

Homma's study [5]:

In his classic study Homma published his findings which suggests that middle ear ossicle resonances for air and bone conduction are slightly different. Measurements of ossicle resonances demonstrated that they show two modes of vibration.

Mode 1: This mode is the primary mode for air conduction. The peak occurs around 1200 Hz. This vibration is caused by hinging movement of ossicles due to air conduction stimulus at the level of umbo of

ear drum.

Mode 2: This mode has a peak around 1700 Hz. This is caused by pivoting motion of malleus and incus complex. This mode is less robust when compared to that of Mode 1 but is dominant one during bone conduction of sound. Decreased mobility of ossicles in this mode caused due to otosclerosis is considered to be the cause for carhart's notch.

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Tondroff hypothesis[6]:

When skull is vibrated by bone conduction, sound is transferred to cochlea via three routes. i.e.

1. By direct vibration of skull

2. By vibration of ossicular chain which is suspended within the skull

3. By transmission via external auditory canal (normal route)

In conductive hearing loss routes 2 and 3 are affected, but can be regained following successful stapes surgery. Hence bone conduction thresholds improve around 2 KHz frequency range.

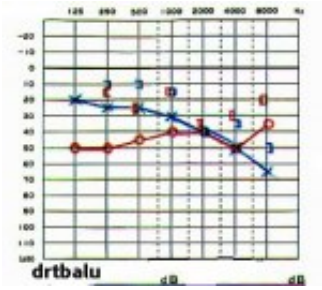

Figure showing carhart's notch

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Blow out fracture orbit Endoscopic reduction

A Novel Management Modality

May 4, 2012 · *Rhinology*

Authors

Balasubramanian Thiagarajan

Abstract

Blow out fracture of orbit involves fracture of orbital floor without fracture of infraorbital rim. This injury is common from frontal blow to orbit. Frontal blow to orbit causes increased intraorbital tension causing fracture of floor of the orbit (weak point) with prolapse of orbital content into the maxillary sinus cavity. This causes enophthalmos and diplopia. Infraorbital rim is not involved in pure blow out fracture, it is also involved then it should be considered as an impure blow out fracture 3. Entrapment of inferior rectus muscle between the fracture fragments will cause diplopia in these patients. This article discusses a novel endoscopic internal reduction of fractured fragments. Main advantage of endoscopic approach is the lack of facial skin incision. It is cosmetically acceptable.

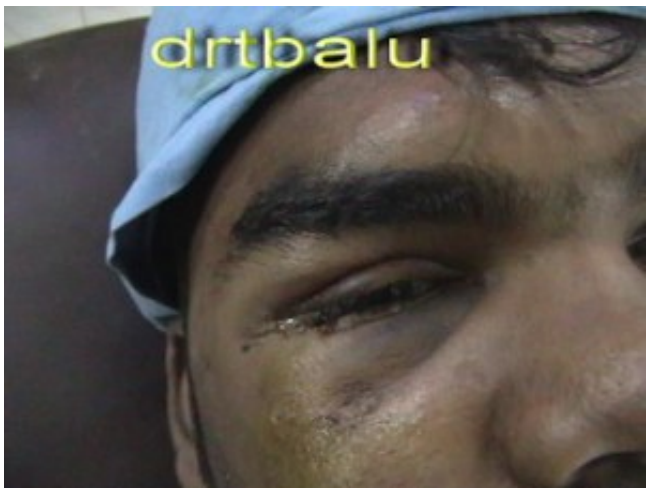
Blow out fracture orbit Endoscopic reduction a novel management modality

Introduction:

Orbital floor fractures were first described by MacKenzie in Paris in 1884 [1]. Smith was the first to describe entrapment of inferior rectus between the fracture fragments. He was also the first to coin the term “Blow out fracture” [2]. Blow out fracture causes an increase in the intraorbital volume, this causes enopthalmos. Entrapment of inferior rectus muscle causes diplopia. These patients usually report to an opthalmologist since orbital signs and symptoms are predominant. Shere etal in their study conclude that nearly 14% of blow out fractures are caused by contact sports in a military population [4].

Case Report:

30 years old male patient came with complaints of:



Clinical photograph of a patient with blow out fracture orbit showing orbital swelling

1. Swelling right eye – 1 day duration

2. Double vision – 1 day duration

3. Bleeding from right nose – 1 day duration

History of injury on being struck by a cricket ball +

He gave no history of loss of consiousness.

On examination:

Swelling over upper and lower eyelids on the right side +

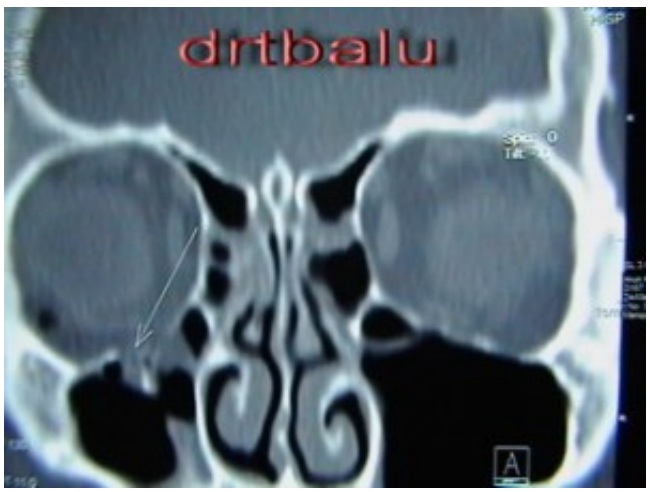
Enopthalmos right eye +

Ocular movements restricted on right gaze

Diplopia +

Forced duction test +

CT scan nose and paranasal sinuses:



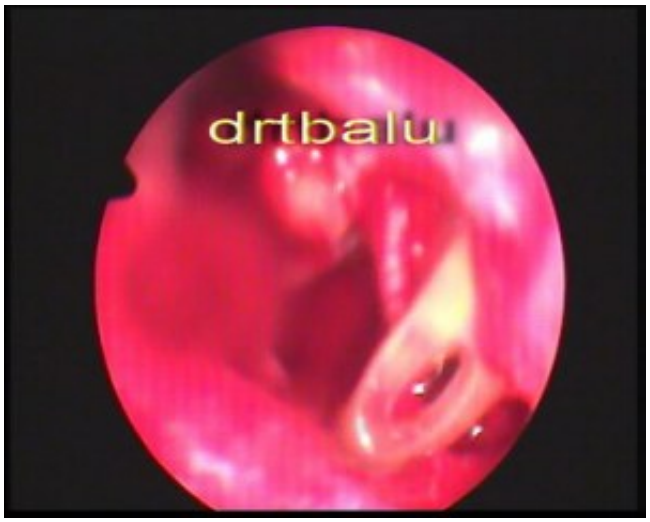
Coronal CT plain of nose and sinuses showing blow out fracture right orbit (classic tear drop sign)

Showed evidence of blow out fracture right orbit. Tear drop sign could be seen.

Management:

Reduction was performed via Caldwel Luc approach under endoscopic guidance. 4 mm 30 degree nasal endoscope was used for this purpose. Trap door fractures can usually be reduced without resorting to prosthesis. Since this patient had a trap door fracture it could be easily reduced under endoscopic guidance. The reduced fracture fragment was stabilized by inflating the balloon of foley's catheter introduced into the maxillary sinus via inferior meatal antrostomy. Foley's catheter is left in place for a period of 2 weeks for union to occur.

Picture showing foley's catheter being introduced into the maxillary antrum via inferior meatal antrostomy



Picture showing inflated foleys catheter inside the maxillary antrum

Discussion:

Orbital blow out fracture is commonly caused by blunt trauma to the orbit. This is commonly seen in persons involved in contact sports like boxing, foot ball, rugby etc [5].

Two theories attempt to explain this injury phenomenon:

1. Buckling theory

2. Hydraulic theory

Buckling theory:

This theory proposed that if a force strikes at any part of the orbital rim, these forces gets transeferred to the paper thin weak walls of the orbit (i.e. floor and medial wall) via rippling effect causing them to distort and eventually to fracture. This mechanism was first described by Lefort 3.

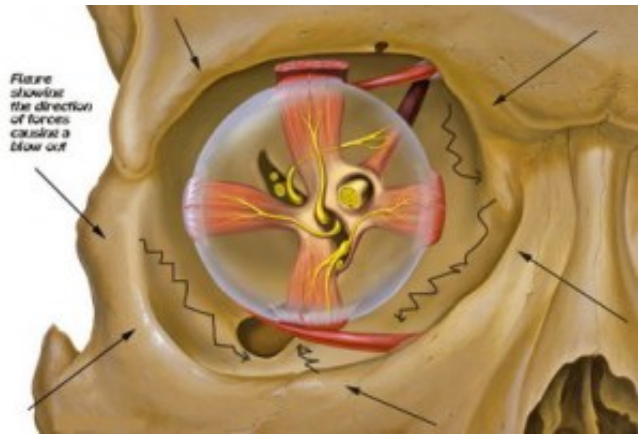
Hydraulic theory [6]:

This theory was proposed by Pfeiffer in 1943. This theory believes that for blow out fracture to occur the blow should be received by the eye ball and the force should be transmitted to the walls of the orbit via hydraulic effect. So according to this theory for blow out fracture to occur the eye ball should sustain direct blow pushing it into the orbit.

Water House [7]in 1999 did a detailed study of these two mechanisms by applying force to the cadaveric orbit. He infact used fresh unfixed cadavers for the investigation. He described two types of fractures:

Type I: A small fracture confined to the floor of the orbit (actually mid medial floor) with herniation of orbital contents in to the maxillary sinus. This fracture was produced when force was applied directly to the globe (Hydraulic theory).

Type II: A large fracture involving the floor and medial wall with herniation of orbital contents. This type of fracture was caused by force applied to the orbital rim (Buckling theory).



Diagrammatic representation of Buckling theory

Initial signs and symptoms of blow out fracture include:

1. Immediate swelling of the eye
2. Tenderness over involved orbit
3. Pain and difficulty with eye movements
4. Double vision
5. Enopthalmos
6. Numbness / tingling over lower eyelid, nose, upper lip[8]

Complications of blow out fracture:

1. Herniation of orbital fat into maxillary sinus[9]
2. Orbital emphysema[10]
3. Bleeding into maxillary sinus
4. Entrapment / rupture of ocular muscles
5. Ischaemic muscle contractures[11]
6. Cellulitis
7. Diplopia

Timing for surgical intervention:

This is highly controversial. Some of the authors prefer a waiting period of atleast 2 weeks for the

oedema to resolve before proceeding with surgical reduction of the fracture. Early intervention is indicated only in white eyed blow out fracture which is common in children. In children the bones are flexible and does not break easily but bends. Significant amounts of orbital tissue may get entrapped in between the fractured fragments causing a compromise in their blood supply. This condition is known as the white eyed blow out fracture. These patients should under go immediate reduction. Surgery is indicated if the eye has recessed by more than 2 mm into the ortbit, ocular movements restricted, persistence of diplopia.

Advantages of endoscopic approach:[12]

1. Accurate fracture visualization

2. Incisions are small

3. Facial incisions can be avoided

4. Minimal soft tissue dissection

5. Hospital stay minimized

6. Cosmetically acceptable[3]

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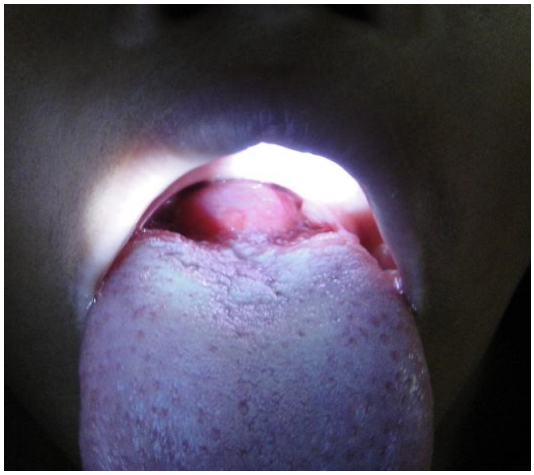


Lingual thyroid & its management II Edition

Dr T Balasubramanian

Lingual thyroid is a rare condition. It is seen roughly 1 in 100,000 populations. Managing this condition is filled with historical controversies ranging from leaving it alone to surgical removal of the lesion. Attempt has been made to present in a precise way the management modalities available. All the surgical modalities along with their pluses and minuses are discussed here.

Balasubramanian Thiagarajan
3/23/2012

ISBN 978-81-923784-1-1

Lingual Thyroid and its management

Balasubramanian Thiagarajan

Publisher: Dr R Geetha
Otolaryngology online

ISBN: 978-81-923784-1-1

Preface

Lingual thyroid is a fairly rare ectopic thyroid presentation. Incidence ranges somewhere in the range of 1 in 100,000 normal populations. Commonly lingual thyroid happens to be the only functioning thyroid. Normal thyroid may be absent in the neck of these patients. Common dilemma in managing these patients is whether to operate lingual thyroid or not. Treatment modality invariably depends on the size of the mass. These patients commonly suffer from hypothyroidism as these ectopic tissues are not capable of maintaining normal thyroxin levels in these patients.

This book discusses etiopathogenesis of lingual thyroid with special emphasis on the various management modalities available.

About the Author

Author is a senior faculty in the department of Otolaryngology Stanley Medical College Chennai India, with rich experience in teaching and training undergraduate and post graduate students. He has also created a few online teaching resource sites for the benefit of students of otolaryngology.

Contents

Introduction:

Lingual thyroid is a rare developmental disorder caused due to aberrant embryogenesis during the descent of thyroid gland to the neck. Lingual thyroid is defined [3] as the presence of thyroid tissue in the midline at the tongue base. It can be present anywhere between circumvallate papillae and epiglottis. Lingual thyroid is the most frequent ectopic location of thyroid gland. Prevalence rates of lingual thyroid vary from 1 in 100,000 to 1 in 300,000 [4]. In nearly 2/3 of these patients lingual thyroid happens to be the only functioning thyroid as normal thyroid tissue is absent in the neck [1]. Review of literature reveals that only about 400 symptomatic cases have been reported so far. This could well be an understatement and statistical anomaly. This condition is 4 times more common in females than males [2]. Even though this condition is diagnosed clinically radio nucleotide scanning is usually confirmatory in nature [3]. Nearly a third of these patients are hypothyroid since the only functioning lingual thyroid cannot cope up with the normal body demands of thyroxin [4]. These patients need to be identified and supplementation should be started at the earliest. Any delay in thyroxin supplementation could lead to enlargement of lingual thyroid tissue causing problems due to mass effect like dysphagia, bleeding from mouth etc.

Literature suggests that ectopic thyroid tissue can occur anywhere, [5] but commonly it is found in midline of neck in the region of hyoid bone, trachea, oesophagus and rarely even in porta hepatis. Histologically majority of lingual thyroid glands demonstrate normal thyroid tissue [17]. Lingual thyroid can be non-encapsulated and could contain embryonic / mature thyroid follicles. This tissue could extend between the lingual muscle fibers [19].

In his classic and extensive study Turot [6] reported that lingual thyroid was present commonly in patients with abnormal thyroid function. He put the figure to be around 1 in 500.

Common locations of ectopic thyroid gland include:

1. Between geniohyoid and mylohyoid muscles (sublingual thyroid)
2. Above the hyoid bone (suprahyoid prelaryngeal)
3. Mediastimum
4. Pericardial sac
5. Heart
6. Breast
7. Pharynx
8. Oesophagus
9. Trachea
10. Lung
11. Duodenum

12. Mesentery of small intestine
13. Adrenal gland

History:
Hippocrates stressed the importance of examination of tongue in diagnosis of diseases. It was Hunt [11] in 1866 first recorded a tumor in the posterior third of tongue.
Hickmann recorded the first case of lingual thyroid in 1869. He reported the death of a 16 hour infant due to asphyxiation caused by goitre at the base of tongue.
Montgomery stressed that for a condition to be branded as lingual thyroid, thyroid follicles should be demonstrated histopathologically in tissues sampled from the lesion. Dore [9] in 1922 collected and analysed 130 cases of lingual thyroid and concluded that majority of them are the only functioning thyroid tissue. He also reported that bleeding from large vessels present over lingual thyroid could be the cause for torrential bleed encountered in these patients.
Bishop [12] practised simple snare removal of lingual thyroid. He suggested snaring lingual thyroid reduced incidence of bleeding. Lahey [13] first classified the various positions the ectopic thyroid tissue could assume.
Montgomery [14] popularised medical management of lingual thyroid by administering Lugol's iodine to these patients. He was able to demonstrate significant reduction in the size of the mass.
Wapshaw [15] popularized extra oral approach to remove large lingual thyroid masses.
It was Thompson who first used diathermy in removing lingual thyroid mass. He was able to demonstrate significant reduction of bleeding during this procedure. Goetsch condemned [16] the use of cautery / radiation in managing lingual thyroid masses.
Lemon and Paschal stressed the importance of neck exploration to ensure normal thyroid tissue is present before proceeding to surgically extirpate lingual thyroid mass. They were also the first to lay down definite indications for surgical removal of lingual thyroid. Lemon after extensive studies concluded that the size of the lingual thyroid mass is an indication for surgical removal of the mass. Other indications suggested by them include:

1. Dysphagia
2. Dysphasia
3. Bleeding from lingual thyroid mass
4. Malignant transformation

Ray and Wapshaw were the first to attempt transplantation of excised lingual thyroid tissue beneath the rectus muscle. Even though their attempt failed, the concept caught the imagination of others who followed them.
Feitelberg was the first person to use radioactive iodine in the management of lingual thyroid mass.

Embryology:
A brief discussion of embryology of thyroid gland will not be out of place as this would ensure better understanding of the pathophysiology involved in the formation of ectopic thyroid gland. Thyroid gland is the first endocrine gland to develop [11]. Its development begins on the 24th day of embryo.
Initially thyroid gland appears as proliferation of endodermal tissue in the floor of the pharynx between tuberculum impar and hypobranchial eminence (this area is the later foramen caecum). Cells of thyroid gland descend into the mesoderm above aortic sac into the hypopharyngeal eminence (later pharynx) as cords of cells. During this descent thyroid tissue retains its communication with foramen cecum. This communication is known as thyroglossal duct. This duct disappears as soon as the descent is complete.
Thyroid gland descends in front of the hyoid bone and laryngeal cartilages [7]. By 7th week it reaches its final destination in front of trachea. At this time a small median isthmus develops connecting the lobes of thyroid gland. The gland begins to function by the 3rd month when thyroid follicles start to develop. Parafollicular or c cells that secrete calcitonin are developed from ultimobranchial bodies.

Persistence of thyroglossal duct even after birth leads to the formation of thyroglossal cyst. These cysts usually arise from the remnants of thyroglossal duct and can be found anywhere along the migration site of thyroid gland. They are commonly found behind the arch of hyoid bone. Important diagnostic feature is their midline location.

Normal development and migration of thyroid gland needs an intact Tbx1-Fgf8 pathway [8]. This pathway has been identified as the key regulator of development of human thyroid gland. Tbx1 regulates the expression of Fgf8 in the mesoderm, it is postulated that Fgf8 mediates critical Tbx1-dependent interactions between mesodermal cells and endodermal thyrocyte progenitors.
Tbx1 is not expressed by thyroid primordium, but is strongly expressed by the surrounding mesoderm. It is also expressed by pharyngeal endoderm lateral to thyroid primordium.
Thyroid organogenesis associated with the expression of a set of transcription factor encoding genes. They include Nkx2-1, Foxe1, Pax8 and Hhex1 genes. Expression of these genes in thyroid primordium is also dependent on Tbx1 gene expression.

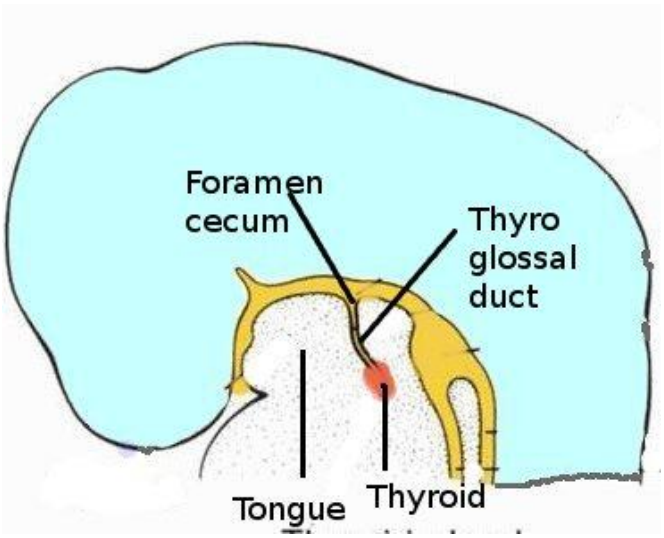


Figure showing development of thyroid ventral to foramen cecum

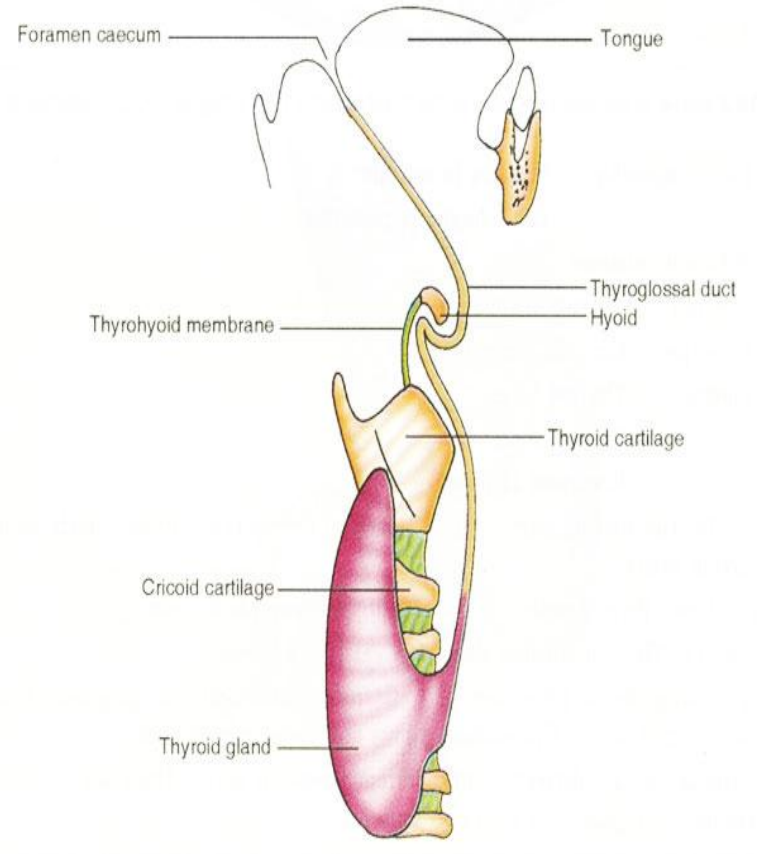


Figure showing migration of thyroid gland

It commonly occurs in females. Female: Male ratio is 4:1. Some studies even attribute it to be 7 times more common in women [17]. Even though lingual thyroid may manifest at any age it is commonly seen in patients in whom there is extra demand of thyroxin by the body which causes it to undergo physiological enlargement. It is commonly seen during early childhood and teens.

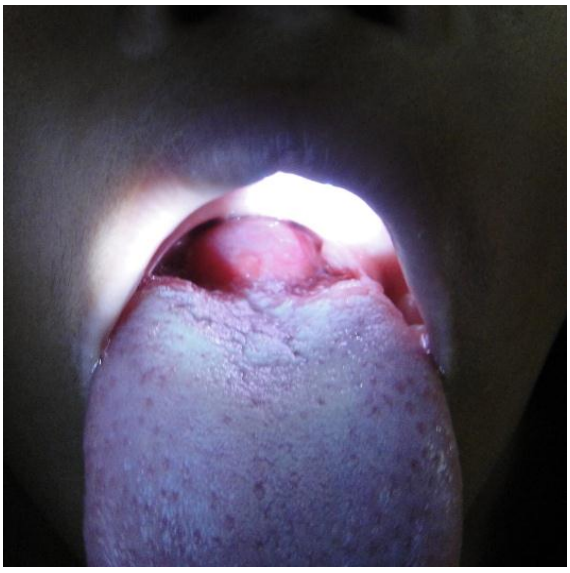
Symptoms:

Majority of these patients are asymptomatic. They will have no problems other than swelling in the posterior portion of their tongue.
Symptoms caused by lingual thyroid include:

1. Dysphagia
2. Dysphonia
3. Bleeding from the mass
4. Sleep apnoea
5. Hypothyroidism
6. Dyspnoea (rarely)

In rare cases lingual thyroid could undergo malignant transformation.

Features seen on examination:



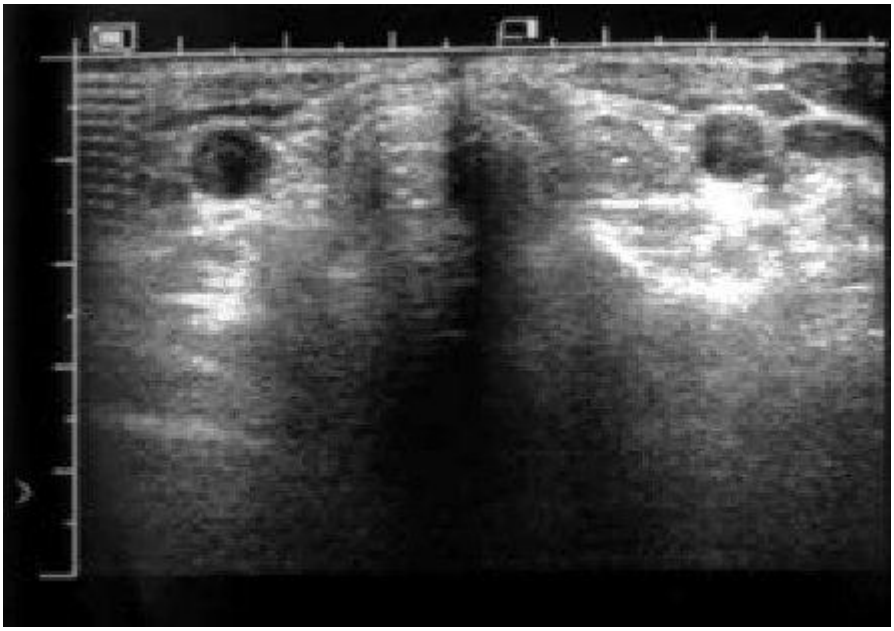
Clinical photograph showing lingual thyroid mass

Table showing symptoms produced by ectopic thyroid tissue according to their location

Location	Symptoms
Lingual	Dysphagia, bleeding, dyspnoea
Suprahyoid / Infrahyoid	Midline neck mass
Thyroglossal duct / cyst	None / Midline neck mass
Pyramidal lobe	None
Intratracheal / Intralaryngeal	Stridor
Intraoesophageal	Dysphagia
Aortic / pericardium / cardiac	None

Lingual thyroid could be seen as pinkish mucosa covered mass over the posterior third of tongue. On palpation this mass could be felt as solid firm and fixed mass. It would be seen attached to the tongue at the junction of anterior 2/3 and posterior 1/3. This is where approximately foramen cecum is supposed to be present. Attempt should be made to palpate the neck in the region of thyroid to ascertain whether normal thyroid tissue is present in the neck

Investigation:
Ultrasound neck:
In all patients with lingual thyroid the presence of normal thyroid in the neck should be ascertained. This can easily be done by performing ultrasound examination of neck. It will reveal the presence or absence of normal thyroid gland in the neck.

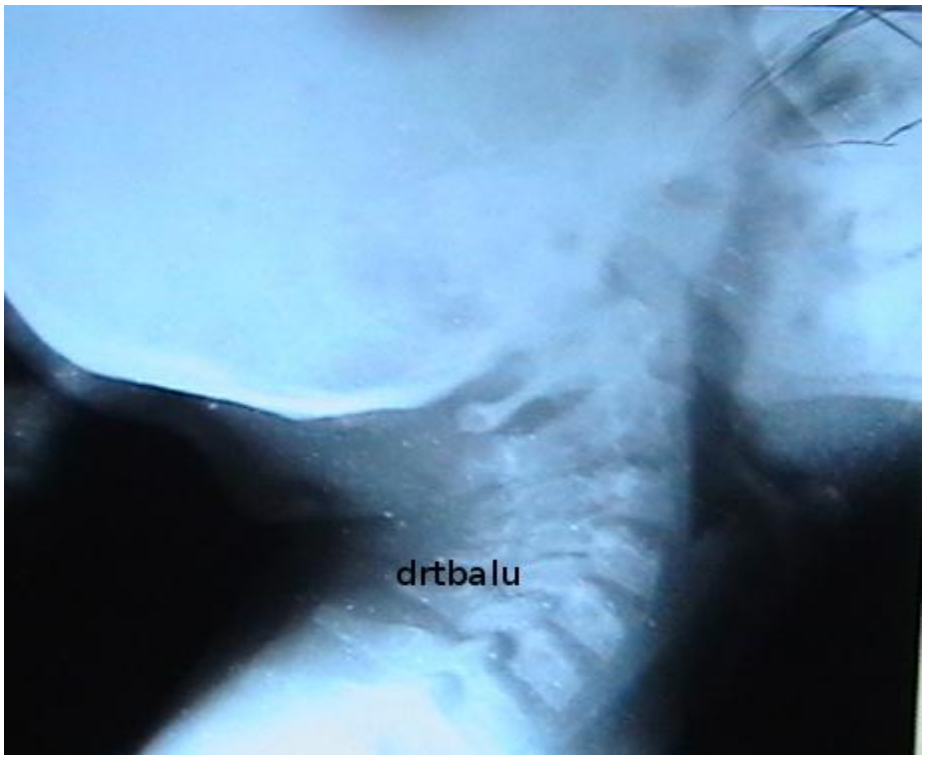


Picture showing ultrasound neck with absence of thyroid gland in the neck

Doppler images reveal peripheral blood vessels and low resistance arterial blood flow. Trans oral ultrasound reveals mass lesion in the posterior third of tongue. It also reveals areas of tissue necrosis if present.

X-ray soft tissue neck lateral view:

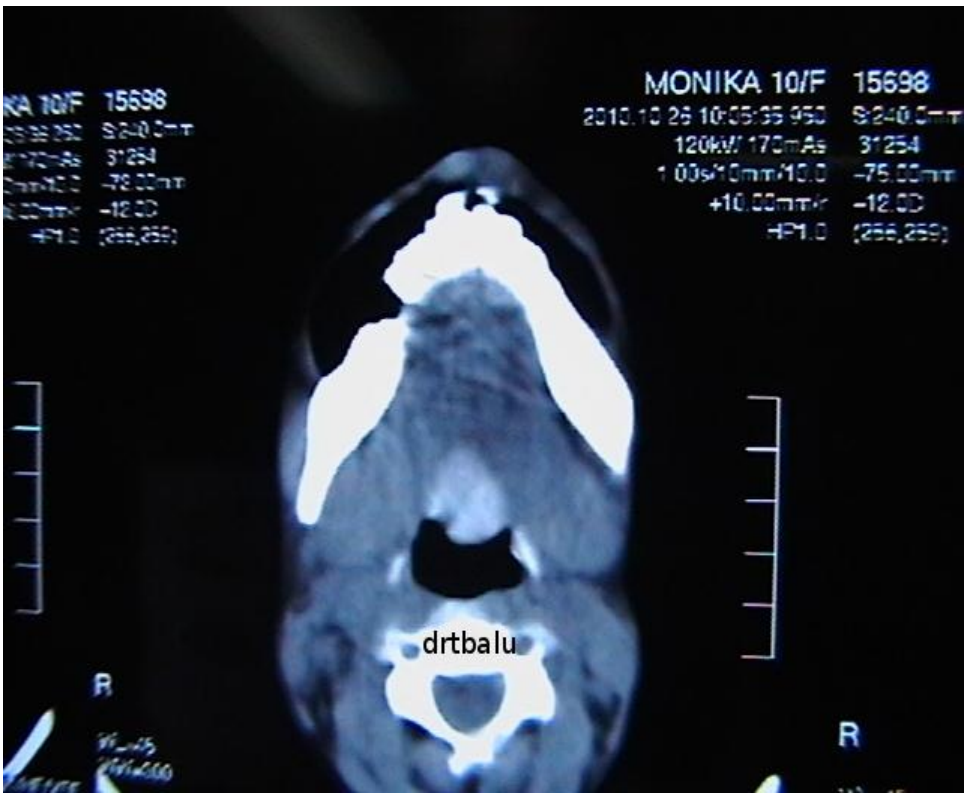
This will just reveal the presence of soft tissue shadow in the region of the tongue. It will also demonstrate the lower extent of the mass.



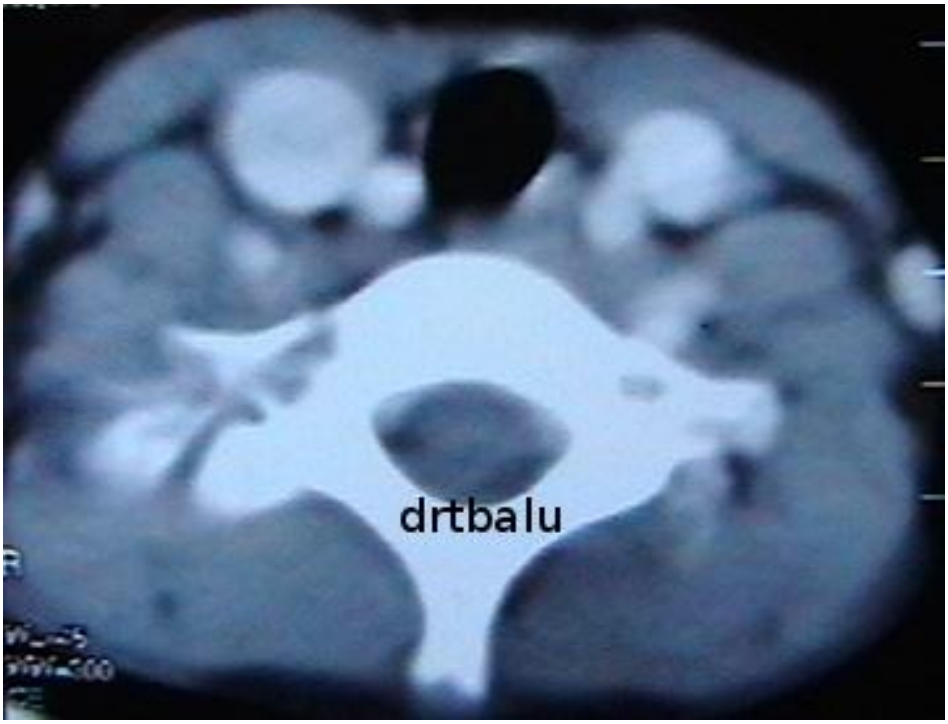
X-ray soft tissue neck lateral view showing a globular soft tissue mass in the region of tongue above the level of hyoid bone

CT scan:

This will help in accurately assessing the extent of lesion. If contrast is used it would give valuable input regarding its vascularity. CT scan of neck will also categorically reveal the presence or absence of normal thyroid tissue in the neck.



CT scan axial cut taken at the level of lower border of mandible clearly shows soft tissue mass occupying the posterior portion of tongue.



CT scan neck axial view with contrast shows absence of thyroid gland in the neck. The internal jugular vein and carotid artery could be seen as enhancing masses. Jugular vein of one side appears to be predominantly enlarged.

Technitium 99 scan is virtually diagnostic. It will clearly reveal the radioactive isotope uptake by the thyroid tissue present on the tongue. It will also clearly demonstrate the presence or absence of thyroid tissue in the neck region. These images are obtained in either dynamic or static mode 20 minutes after intravenous injection of 74-111MBq of Technitium 99 pertechnetate. Its molecular weight is comparable to that of iodine and is transported actively into the thyroid tissue via the sodium iodide symporter system.

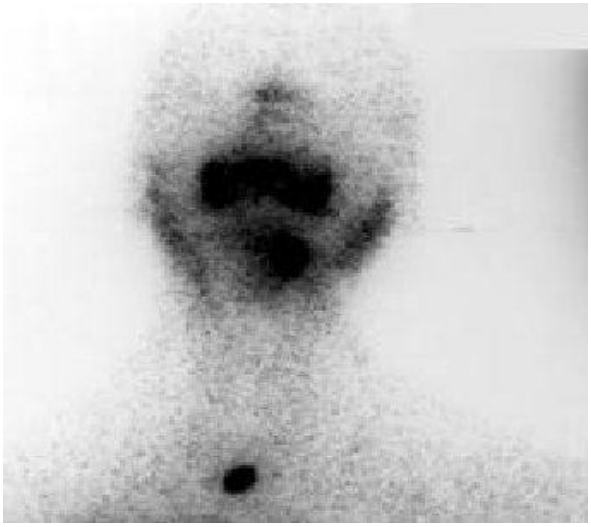


Figure showing Technitium 99 scan. It clearly shows increased uptake in the region of the tongue (due to lingual thyroid tissue) and absence of uptake in the neck region due to absence of normal thyroid tissue in this area. It also helps in location of ectopic glands [18].

Role of radio active iodine uptake studies:

This helps in ascertaining the functional status of the thyroid gland. It also helps in ascertaining the viability of the transplanted ectopic thyroid gland 100 days after the surgical procedure.
Both I 131 and I 123 can be used for this purpose. I 123 have a favourable dosimetry for imaging. Since it is produced in a cyclotron it is rather expensive. Whereas I 131 is reactor produced and is reasonable cheap. It is also freely available. It has poor imaging characteristics and emits beta radiation. Its half life is about 8 – 10 days as compared to 12 hours of I 123. Hence I 123 is preferred for functioning radioactive imaging purposes.
Radioactive iodine is usually administered in small doses orally and uptake is measured at different intervals i.e. 2 hrs, 4 hrs, 24hrs and 48 hrs.

Estimation of serum T3 T4 and TSH levels:

This will help in assessing the functional status of the ectopic gland. Invariably

majority of these patients are euthyroid. If TSH levels are raised then suppression can be attempted using regular doses of oral thyroxine.

Management:

Conservative: If the lingual thyroid is the only functioning thyroid suppression therapy using regular oral doses of thyroxin can be attempted. This is more so in patients whose normal physiological requirement of thyroxin is raised as during periods of active growth, menarche, pregnancy etc. This suppression therapy will help in preventing abnormal physiological enlargement of the ectopic thyroid tissue.

Surgical management:

Indications for surgery:

1. If the mass produces obstructive symptoms
2. If the mass produces bleeding
3. If the mass demonstrates sudden increase in size
4. If malignancy is suspected

FNAC is not advised as it would cause unnecessary bleeding. Similarly instead of biopsying the lesion total excision is preferred.

Methods of excision:

Transoral method of excision:

This method of excision is preferred for small lingual thyroid masses. It is ideally suited for lesions which are above the level of hyoid bone. Clinically if the posterior border of the swelling is seen on depressing the tongue with a tongue depressor then one can safely go ahead and remove the mass transorally.
Transoral removal is assisted by:

1. Cautery
2. Coablation
3. Debrider
4. Laser

Surgery is usually performed under general anesthesia induced via nasotracheal intubation. This is the preferred intubation modality in these patients as it would avoid troublesome bleeding following intubation trauma.
Patient is placed in Rose position. Boyles Davis mouth gag is used to hold the mouth

open. Throat is packed tightly using ribbon gauze to avoid spillage into larynx. The mass is held with a tenaculum forceps and is pulled anteriorly. The anterior border is incised using diathermy cautery / coblator /laser. The tumor is gently dissected and stripped away from the lingual tissue. Perfect hemostasis is secured by coagulating the bleeding points seen in the base of the tumor.

Debrider blade can be used to shave off the tumor from the tongue base. Bleeding points seen in the base can be cauterized using bipolar cautery.

Advantages of transoral approach:

1. Easy to perform
2. Neck incision is avoided
3. Patient's recovery is rapid
4. Complications are minimal

Transmandibular translingual approach:

This approach is very useful in removing very large lingual thyroid masses.

Procedure:

Preliminary tracheostomy is performed under local anesthesia. General anesthesia is introduced via tracheostome. This protects and takes control of the airway in an efficient manner.
An incision over the mucoperiosteum of the buccogingival sulcus is performed over the interior region of mandible and the bone over the mental area is exposed. A midline vertical osteotomy of the mandible is performed. The tongue is sectioned sagittally in the midline up to the floor of the mouth till the tongue base is reached. The lingual thyroid mass is excised in toto. The wound is closed in layers. The mandible is immobilized by wiring and dental arch bar.

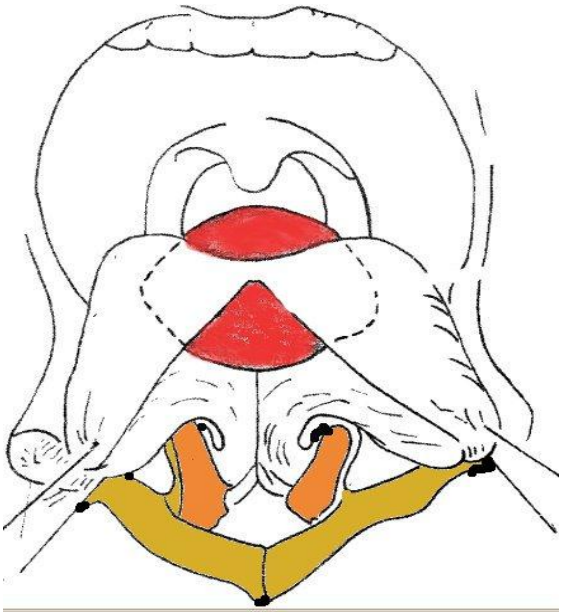


Figure showing the transmandibular approach

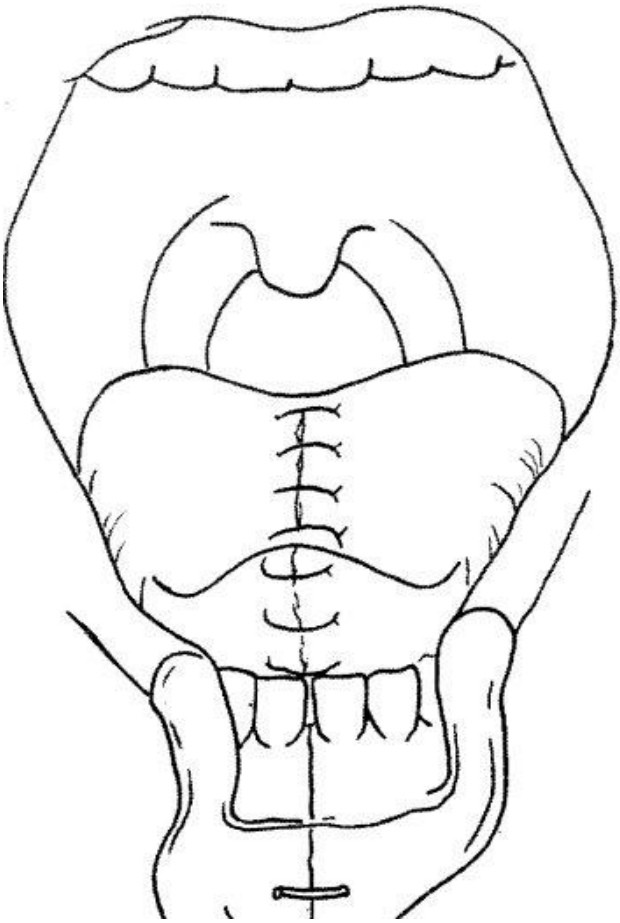


Figure showing wound closure

Advantages:

1. Excellent visualization
2. No need for ligating lingual vessels
3. Important structures are spared i.e lingual nerve, hypoglossal nerve, and submandibular salivary gland

Lateral pharyngotomy approach:

This approach is preferred if transpositioning of lingual thyroid is planned. Anaesthesia is induced via nasotracheal intubation. Patient is positioned in such a way that the neck is slightly extended. An oblique curved incision is made about 8 cms long in the left lateral portion of upper neck just anterior to sternomastoid muscle. The dissection is proceeded in the subplatysmal plane. The following structures are identified:

1. Carotid bifurcation
2. Lingual artery
3. Superior thyroid artery
4. Hypoglossal nerve

Using the finger guide passing through the oral cavity to the left lateral pharynx at the level of base of tongue a lateral transverse pharyngotomy of 3-4 cms is made inferior to the hypoglossal nerve and above the hyoid bone.
Through this pharyngotomy opening the posterior 1/3 of tongue, epiglottis and lingual thyroid mass could be identified. The gland is dissected out of the tongue. The right side of the mass is totally freed of the tongue. The mass is mobilised by an encircling incision over the tongue. A small attachment to the left side of tongue base is retained. This will ensure adequate vascularity to the mass after transposition. The mass is delivered via the pharyngotomy opening and is implanted in the left side of the neck with its attachment to the left tongue base remaining intact. The wound is closed in layers.

Advantage:

The most important advantage of this approach is that it ensures tension free transposition of lingual thyroid to the left side of neck. After transposition the gland can easily be examined on the left lateral neck of the patient.

Suprahyoid midline approach:

This approach is preferred in removing large lingual thyroid mass even if it extends to a level below that of hyoid bone.

Procedure:

This surgery is performed under general anesthesia administered via nasotracheal intubation. This intubation modality prevents intubation injury to lingual thyroid mass.

Infiltration:

The surgical area in the neck is liberally infiltrated using tumescent fluid.

Tumescent fluid is prepared using:

1. one litre of ringer lactate solution
2. 40 ml of 2% xylocaine
3. 1ml of 1 in 1000 adrenaline
4. 20 ml of 8.4% soda bicarb

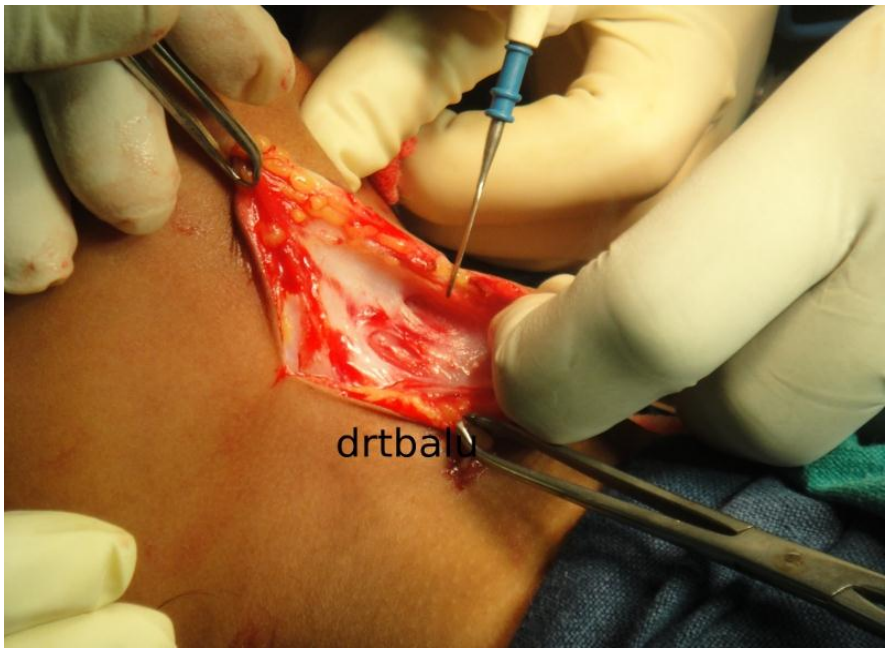
Advantages of using tumescent fluid infiltration:

1. Breaks open tissue planes facilitating easy dissection i.e Hydro dissection
2. Reduces bleeding due to vasoconstrictive effect of adrenaline
3. Facilitates uniform heat dissipation when diathermy is used during surgical procedure
4. Prevents development of local tissue level acidosis

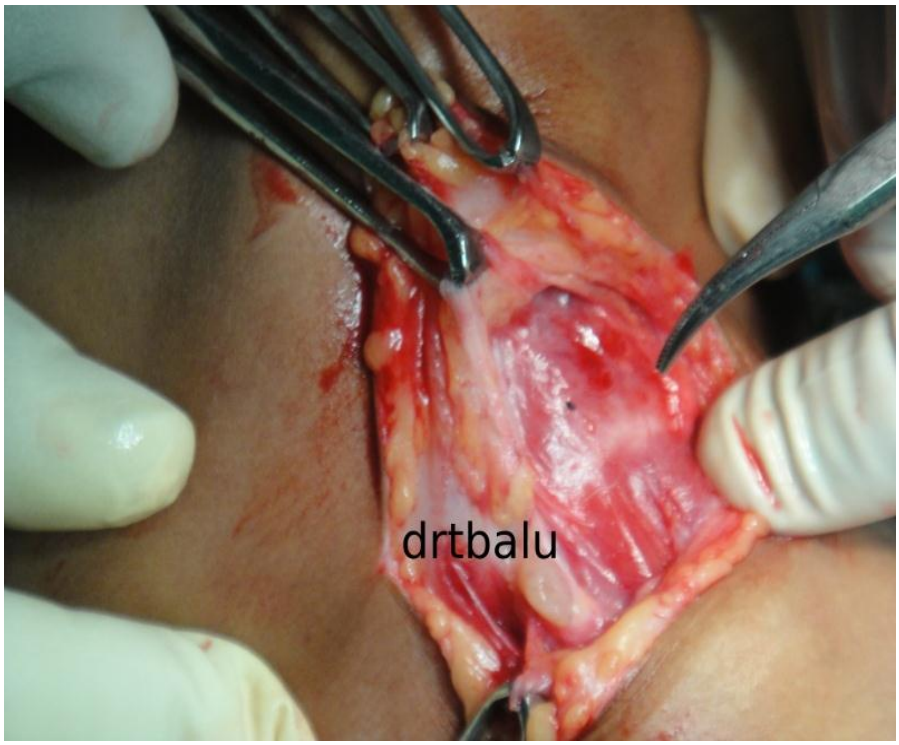
Figure showing infiltration being given

Incision:

Transverse skin crease incision is made at the level of hyoid bone. Skin, subcutaneous tissue and cervical fascia are elevated in the subplatysmal plane. Sticking on to the subplatysmal plane helps in preserving the cervical branches of facial nerve. Dissection in this plane is continued and the flap is raised above the level of hyoid bone.



Incision being widened using cutting diathermy



Hyoid bone visualized

Supra hyoid dissection:

In this stage the muscles attached to the hyiod bone are cut and dissected subperiosteally.

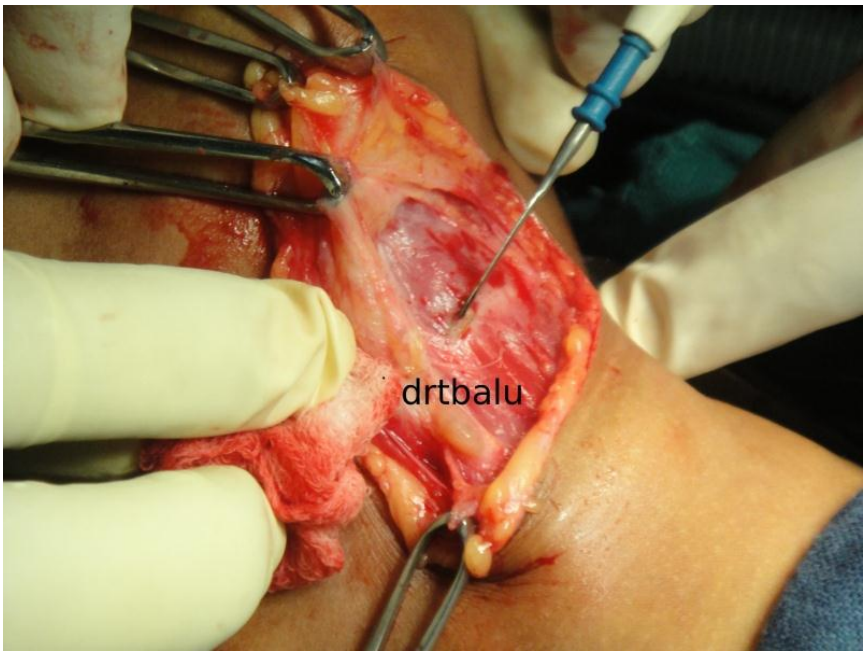


Figure showing hyoid bone being skeletonized using a cutting diathermy

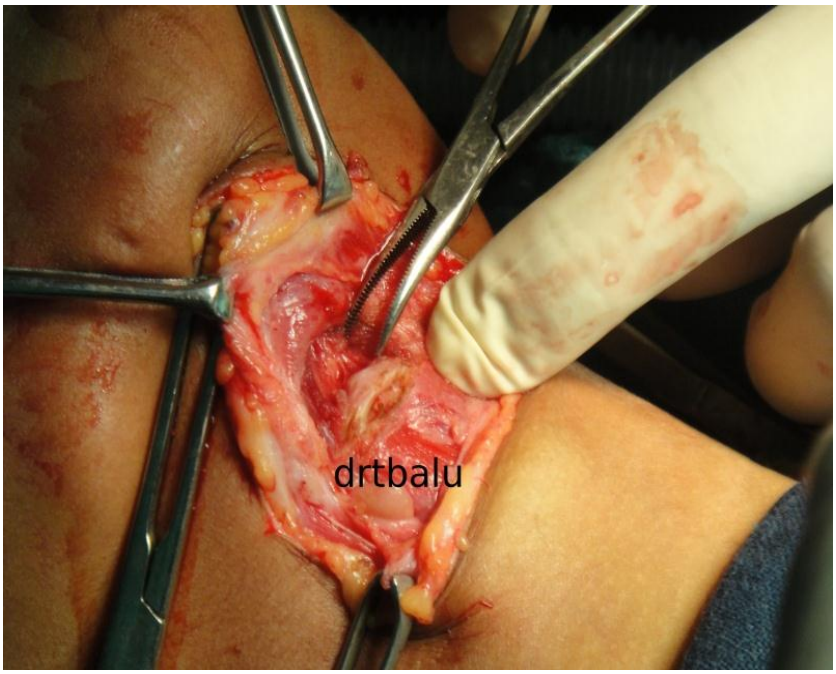


Figure showing suprahyoid subperichondrial dissection being performed

The supra hyoid muscles are split and the oral cavity is entered. Using a finger guide inside the oral cavity the mass is pushed downwards and delivered via the suprahyoid neck incision. The mass is removed in full. The wound should be meticulously closed in layers. Ryle's tube should be inserted to facilitate early feeding. Ideally the Ryles tube should be left in place at least for 3 days.

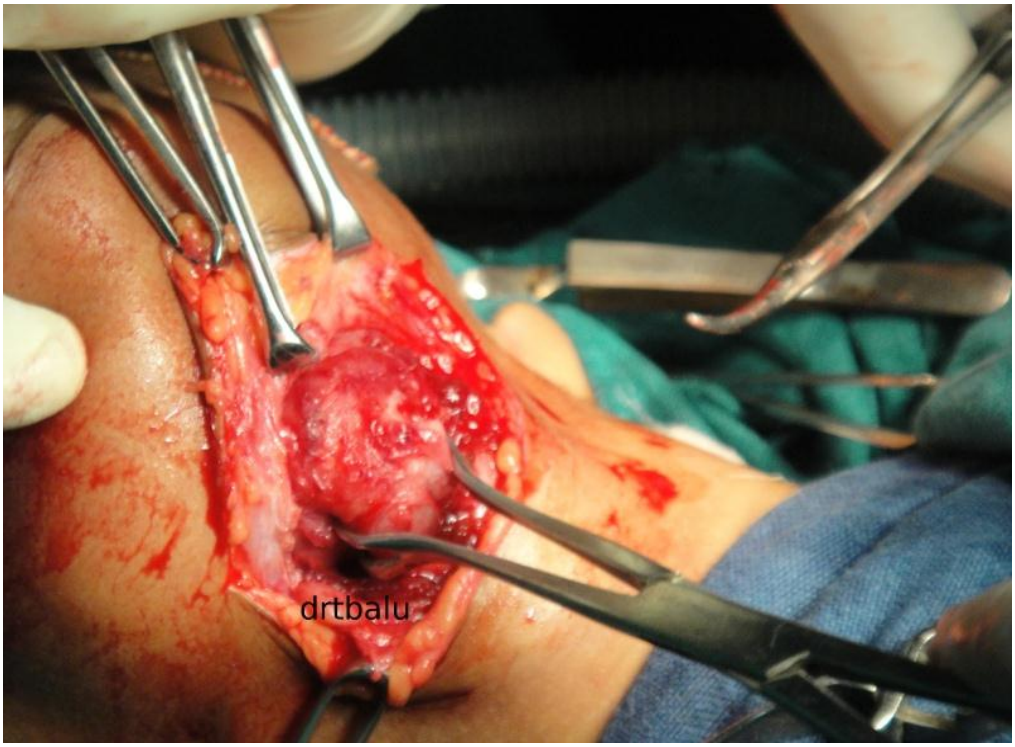
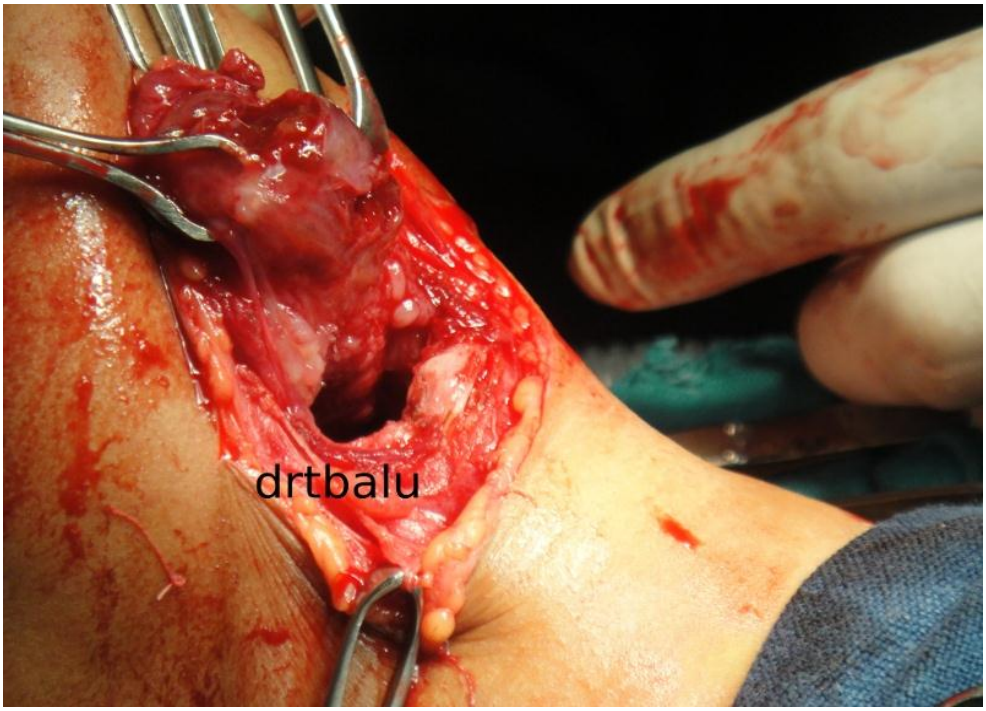


Figure showing lingual thyroid being delivered in to the neck



Lingual thyroid attached to the base of tongue

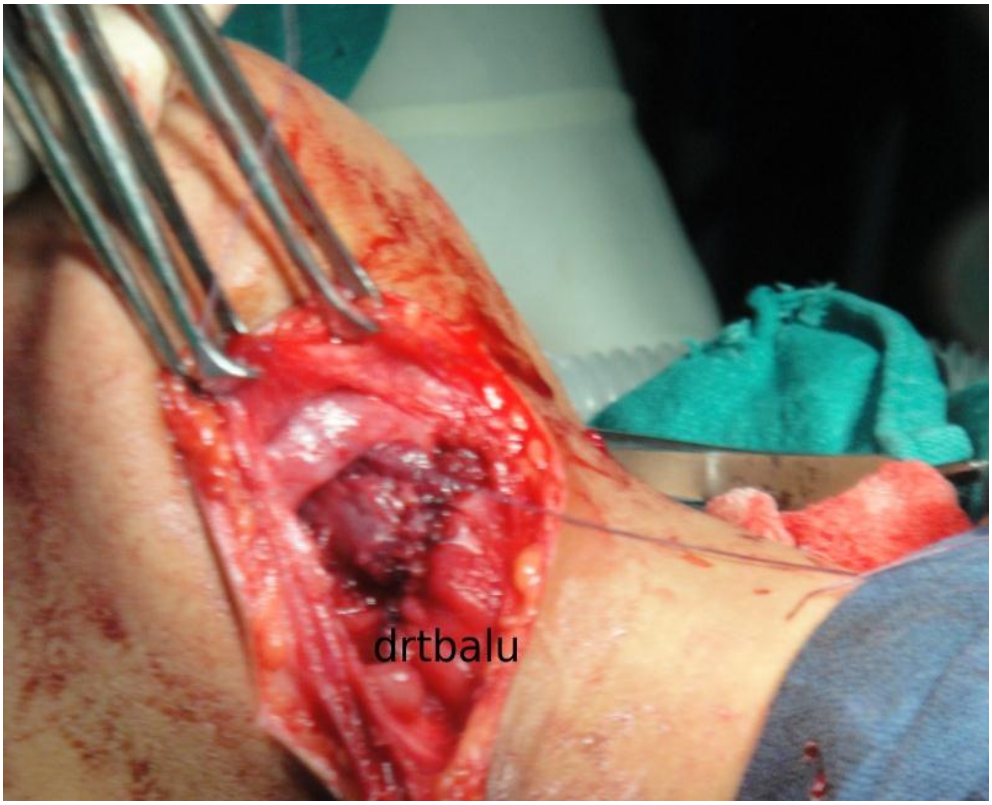


Figure showing wound closure

After surgery all these patients should be started on oral supplemental doses of thyroxin.

If you are wondering about the status of parathyroids, you need not worry as they will be in their normal position i.e. neck because embryologically their developmental process is different.

Radiofrequency ablation [20]: This has been successfully used in managing lingual thyroid masses. Bleeding is minimal and complete removal is possible with minimal morbidity and tissue damage. Lingual oedema is also minimal in these patients.

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